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RHINOGENIC RETROBULBAR NEURITIS.

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The reports concerning the frequency of rhinogenic retrobulbar neuritis vary considerably. While Grosz[27] finds a rhinogenic etiology in 15 per cent, Bachstez[2] in 13.7 per cent of cases of retrobulbar neuritis, Hajek[29] considered this origin as rare, and Cushing[18] apparently doubted its existence in all instances. As a matter of fact, there are clinicians today who consider rhinogenic retrobulbar neuritis as a rarity and even deny its existence. Consequently, an investigation as to whether or not a rhinogenic retrobulbar neuritis actually exists seems to be justified. Inasmuch as Van der Hoeve[37] and others have emphasized that there is no clear-cut eye symptom at the disposal of the ophthalmologist indicating a retrobulbar neuritis of rhinogenic origin, the rhinologist must perforce discern the signs leading to such a diagnosis. These signs concern: *1.* The anatomy, *2.* the treatment, *3.* the pathology of so-called rhinogenic retrobulbar neuritis.

I. ANATOMICAL CONSIDERATIONS.

Onodi,[55] Loeb[48] and, more recently, Climelli (quoted by Salinger[60]) and Van Alyea[1] have made exhaustive studies concerning the anatomical relationship between the optic nerve and the posterior sinuses. Onodi[55] found 38 different morphological conditions bearing on the relation between these two organs. All of these investigations led to the following conclusions:

1. Only the posterior sinuses, *viz.*, the sphenoid and the posterior ethmoids, frequently show an intimate relation to

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the optic nerve. Consequently, all cases in whom the retrobulbar neuritis followed an inflammation of the maxillary or the frontal sinus must be regarded as doubtful. It is true that Onodi[55] mentions that the ethmoid cells might be represented by a cavity which is merged with the frontal sinus and thus forming one common cavity. However, this finding is so rare that for practical purposes it does not come into consideration. Nevertheless, Nakamura (quoted by Salinger[60]) reports two cases who were cured by treatment of the maxillary sinus. It is questionable whether or not the author observed the cases over a sufficiently long period to ascertain that the retrobulbar neuritis was actually due to the infection of the maxillary sinus. Cullom[19] reports a case in whom the vision was markedly improved by an operation on the maxillary sinus. Despite this result, Cullom considers his case not as of rhinogenic origin but rather as a toxic amblyopia (which is so frequently the case). *2.* The sphenoid or the posterior ethmoid sinuses may have an intimate connection with the optic nerves on both sides. This finding explains the occurrence in instances of unilateral infection of the posterior sinuses and a bilateral or a unilateral retrobulbar neuritis on the side opposite to the infected sinus. *3.* The wall between the posterior ethmoid cells and the optic canal is usually as thin as paper, while the thickness of the wall between the sphenoid and the optic canal varies between 1-12 mm. Consequently, an inflammation of the posterior ethmoid cells is, as far as the optic nerve is concerned, at least as dangerous as is that of the sphenoid. *4.* Bony dehiscences of the optic canal may facilitate or cause orbital disturbances. However, such dehiscences are rare. Onodi[55] found them once among 300 specimens, Galmaerts (quoted by Beck[4]), twice in 290 specimens, and Van Alyea[1] did not find any dehiscences among 100 specimens. Furthermore, the pathology of the temporal bone has proved that dehiscences of the tegmen tympani are not as dangerous as was believed in the past, in so far as the passage of infection is concerned. *5.* If the infection originating in the posterior sinuses actually invades the optic canal, it must first meet with the canalicular portion of the optic nerve. Since it usually takes a considerable period of time for the infection to descend into the region of the bulb, it is obvious that in the incipient stage of a retrobulbar neuritis there are no pathologic findings in the bulbus oculi.

To sum up, all these investigations prove one fact; *viz.*, that there is frequently only a very thin bony wall between the sheaths of the optic on the one hand, and the mucous membrane of the posterior sinuses on the other. As Van der Hoeve[32] has emphasized, we usually take it for granted that the danger for the nerve is the greater the nearer it is to the diseased sinus. This assumption would be correct if rhinogenic retrobulbar neuritis were due to a destruction of the bony wall and the subsequent development of a fistula between the sinus and the optic canal.

Some rhinologists actually hold this opinion; for example, Tunis,[66] v. Bajkay[3] and others. However, this is not the pathology of retrobulbar neuritis, but it rather could be the pathology of an optic neuritis associated with a phlegmon of the orbit or a meningitis (Haskin[30]). As far as I know, never was there found a fistula in the wall of the sphenoid or the posterior ethmoid in simple retrobulbar neuritis, unless the latter was due to a malignant tumor of the posterior sinuses. In instances of simple retrobulbar neuritis of rhinogenic origin the invasion of the optic canal seldom (probably never) is due to a phlegmonous inflammation with the development of a fistula. Consequently, the infection reaches the optic nerve solely along the pathways of connective tissue strands, nerves or blood vessels. In such a case it does not make much difference whether the thickness of the bony wall is 1 or 12 mm., inasmuch as the soft tissues are of major importance, as emphasized by Van der Hoeve,[37] Gradle,[26] Wherry[70] and many others.

As a matter of fact, in cases of rhinogenic retrobulbar neuritis but occasionally a particular size or form of the sinus was observed, as, for instance, in the case of Elles,[21] in whose patient a complete partition of the sphenoid was found, below which was a large cell extending into the pterygoid process.

The question which now arises is the following: Is there soft tissue which connects the mucous membrane of the sinus with the sheaths of the canalicular portion of the optic nerve? As far as lymphatics are concerned, they cannot be considered as pathways since Gruenwald[28] has shown that the lymphatics of the mucous membrane of the paranasal

sinuses do not perforate the bone and thus cannot reach the orbit.

The blood vessels, the arteries of the orbit, as well as the veins, are in communication with the respective blood vessels of the mucous membrane of the paranasal sinuses. The canalicular portion of the optic nerve is nourished by arterioles originating from the A. ophthalmica within the optic canal. Inasmuch as the A. ophthalmica also gives source to A. ethmoidalis anterior and posterior, it is obvious that the optic nerve and the superior part of the nose receive their arterial blood from the same source.

However, as far as the transmission of inflammatory changes are concerned, the veins are of greater importance. I observed in the lamina papyracea of the newborn an amazing number of small veins originating in the mucous membrane of the nose, perforating the lamina papyracea and the periosteum of the orbit and invading the orbit, where they apparently empty into the Vv. ophthalmicae. Carmi and Pientrantoni[13] presented an exact description of these veins. However, these veins, as far as they communicate with the Vv. ophthalmicae, cannot be considered of importance in the pathology of the retrobulbar neuritis because they have relations only to the intraorbital and not to the canalicular portion of the optic nerve. Then, too, inasmuch as the Vv. ophthalmicae leave the orbit through the superior orbital fissure (occasionally through the inferior orbital fissure) and not by way of the optic canal to reach the cavernous sinus and the pterygoid plexus, respectively, this possibility seems remote. I agree fully with Gradle,[26] who emphasized some years ago that the intraorbital portion of the optic nerve is not apt to be affected by any disease, except a purulent inflammation of the orbit which would be of such intensity as to eliminate any observation of the blind spot. Consequently, the Vv. ophthalmicae, with their contributaries from the ethmoid, cannot be of importance insofar as the pathology of the retrobulbar neuritis is concerned. More important, however, is the V. centralis posterior nervi optici, first described by Vossius.[68] This vein collects the blood from the posterior part of the optic nerve, with its sheaths, and from the periosteum of the optic canal. The vein lies in the exact center of the canalicular portion of the optic nerve, leaves it at the pos-

terior end of the optic canal and pours directly into the cavernous sinus. Furthermore, Uffenorde[67] has shown that tiny veins originating in the mucous membrane of the sphenoid, perforate the optic canal and apparently empty into the V. centralis posterior. On the contrary, Herzog[34] found that one seldom encounters these perforating veins. He rather lays stress upon the marrow spaces situated between the sheaths of the optic nerve and the mucous membrane of the posterior sinuses. Herzog[34] examined the optic canal microscopically and found the following details: The bone of the optic canal has, in general, a moderate thickness, occasionally dehiscences can be seen where the mucous membrane of the sphenoid and the dural sheath of the optic nerve are adjacent. As a rule, the bone is rather compact and reveals only a small number of Haversian channels and tiny marrow spaces. In a smaller group of specimens there were more channels and the marrow spaces were larger and more numerous. Serial sections proved that frequently such marrow spaces open toward the mucous membrane on the one hand, and toward the dura sheath of the optic nerve on the other. Occasionally the bone contains numerous and very large marrow spaces so that the compact bone is changed into a spongy one. Naturally, in such cases the communication between the mucous membrane and the dural sheath is of a greater extent. In such specimens the submucosa invades the marrow space and merges loosely there into loose connective tissue strands. On the other side, parts of the dura penetrate the marrow space and occasionally may even reach the mucosa of the sphenoid sinus. Summarizing, there can be no doubt that blood vessels, as well as marrow spaces, furnish a definite connection between the mucous membrane of the posterior sinuses and the sheaths of the optic nerve.

Haskin[30] has emphasized that sympathetic fibres of the carotid and cavernous plexus conducting trophic fibres for the optic nerve are adjacent to the walls of the posterior sinuses. However, since communicating fibres between these sympathetic plexuses and the mucous membranes are absent, an infection cannot travel from the sinuses into the optic canal along the nerve fibres.

The anatomic considerations prove that there is undoubtedly a connection between the mucous membrane of the

sinuses and the sheaths of the optic nerve, by virtue of marrow spaces containing connective tissue and small veins. However, because there is such an atrium, it does not necessarily hold true that infection must enter in this manner.

II. THERAPEUTIC CONSIDERATIONS.

The most outstanding reason for the concept of the rhinogenic origin of a retrobulbar neuritis was the fact that in many cases the eye symptoms subsided after intranasal manipulations. However, as it often occurs in therapeutic problems, the question as to whether one is concerned with a *post hoc* or a *propter hoc* is very difficult to say. This question is particularly difficult to answer in retrobulbar neuritis, inasmuch as all observers admit: *a.* that disseminated sclerosis is more frequently the reason for a retrobulbar neuritis than are the inflammatory states of the paranasal sinuses: and *b.* that the retrobulbar neuritis occasionally may be the sole symptom of a disseminated sclerosis over a long period of time.

(a)—RETROBULBAR NEURITIS AND DISSEMINATED SCLEROSIS.

A great number of statistics are published to demonstrate the various reasons for the origin of a retrobulbar neuritis. These statistics have produced considerable confusion because the incidence of disseminated sclerosis is estimated to be from about 20 to 80 per cent (Rea[57]), while others, on the contrary, attribute a nasal origin to 70 per cent of the cases.

According to Gifford,[25] this conspicuous confusion may be explained by two facts: *a.* Many observers consider recovery of vision after a nasal operation as definite proof that the retrobulbar neuritis was of rhinogenic origin. *b.* Many clinicians forget, as mentioned above, that occasionally the retrobulbar neuritis may be the only symptom of a disseminated sclerosis over a long period of time. The last-mentioned fact is known for many years. Consequently, cases of retrobulbar neuritis, who after an intranasal operation are not observed over a long period (at least one year), do not prove anything insofar as the etiology is concerned. There are only rare exceptions, which will be mentioned later. However, there must be a limit also in considering disseminated sclero-

sis as etiologic factor. If in a case of retrobulbar neuritis a few months, or even one year after the operation, a disseminated sclerosis is found, it sounds plausible that in such an instance the neuritis was actually a prodromal symptom of the disseminated sclerosis. If, however, many neurologists (Oppenheim, Stern,[64] Brain[11] and others) claim that a retrobulbar neuritis may precede a disseminated sclerosis, say prior to 15 or 20 years, it should be the task of the neurologists to demonstrate a sclerotic focus in the optic nerve or, at least, to prove that in a particular case the vision of the patient remains permanently impaired despite an intranasal operation. One must always bear in mind that there are patients who have a disseminated sclerosis plus inflammatory changes in the paranasal sinuses. In such a case it is very difficult to decide whether the retrobulbar neuritis is due to disseminated sclerosis or the result of nasal pathology. Meller believes that the changes which take place subsequent to a sinusitis prove to be disposing factors in the development of sclerotic foci within the optic nerve. Meller and Hirsch[52] state that the mucous membrane of the ethmoid, even if it appears macroscopically normal, might be the source of an infection of the optic nerve. They base their statement on the experiments of Behr,[7] who produced in rabbits symptoms similar to those of a disseminated sclerosis by implantation of mucous membrane of the ethmoid into the subdural spaces.

At present, the concept of Meller and Hirsch[52] lacks an actual basis. First, the experiments of Behr[7] have not as yet been confirmed. Second, although the evidence appears to be more in favor of the view that disseminated sclerosis is due to a neurotropic infection and is an encephalomyelitis, characterized pathologically by perivascular demyelinization and clinically by its relapsing tendency, it must be admitted that all of the reported experimental evidence which claims to demonstrate the transmission of disseminated sclerosis to animals is open to considerable criticism (Brain[11]).

To sum up, the paranasal sinuses, as well as disseminated sclerosis, might be the etiologic factors in the development of retrobulbar neuritis, but there is no doubt that disseminated sclerosis is of greater importance than the paranasal sinuses, as emphasized recently by Moore,[53] Lillie[47] and Benedict.[8] Inasmuch as but few clinicians doubt this fact, it is useless

to publish statistics in order to again prove it. However, neurologists should not exaggerate the neurogenic factors, just as in the past the rhinologists have overemphasized the rhinologic etiology. Consequently, if Brain[11] and others contend that every case of retrobulbar neuritis for which no other cause can be found should be regarded as a possible case of disseminated sclerosis, we would rather agree with the viewpoint of Gifford[25] that it may prove to be equally erroneous considering all cases of retrobulbar neuritis in which empyema of the sinuses cannot be demonstrated as due to disseminated sclerosis, even in the absence of all other signs pointing to it.

(b)—EVIDENCE OF THE RHINOGENIC ORIGIN OF RETROBULBAR NEURITIS.

At first, we wish to recall these cases of retrobulbar neuritis which were cured by intranasal treatment and were observed for a period of from six to 10 years without showing any signs of a retrobulbar neuritis or disseminated sclerosis. Such cases were published by Stenger,[63] Oliver and Crowe,[54] Bachstez[2] and many others. We further wish to reiterate the clinical observations which seem to prove that the blood vessels of the optic nerve can be influenced by way of the nose. De Kleyn[40] and Bordley[10] placed tampons over the sphenoid ostium. Two of the eight cases of Bordley[10] showed field changes following this procedure, one developing a marked increase in the blind spot and a relative scotoma five hours later, which cleared up when the tampon was removed. Krassnig[45] enlarged the sphenoid ostium in a case of retrobulbar neuritis. The eye symptoms immediately recurred when the sphenoidal ostium was closed by granulations and then disappeared when a further enlargement of the ostium was performed. Carmi and Pientrantoni[13] observed a pallor of the disc after application of cocaine plus adrenalin to the mucous membrane of the nose. Forschner and Sommer[22] reported a case of ozena in whom a retrobulbar neuritis occurred two days after an injection of ozena vaccine into the middle turbinate. Finally, one must recall those cases which are fortunately rare, who acquire a transitory amaurosis, with or without changes in the disc, after the injection of novocaine into the mucous membrane of the nasal septum.

Although Pichler[56] found that in animals, corpuscular elements travel readily from the paranasal sinuses into the orbit, one must rather assume that the described changes of the optic nerve occur by a trigeminosypmpathetic reflex by way of the sphenopalatine ganglion, as emphasized by Haskin, Escat and Sourdille. Even the most critical consideration of the therapeutic end-results points to the fact that a rhinogenic retrobulbar neuritis actually exists.

III. PATHOLOGIC CONSIDERATIONS.

While the anatomical as well as the therapeutic considerations have led to the conclusion that a rhinogenic retrobulbar neuritis undoubtedly exists, the pathologic considerations supposedly support this conclusion. Unfortunately, the pathologic considerations fail to do this, inasmuch as there is an insufficient number of cases on record who were examined postmortem.

Considerable effort was given to the microscopic examination of tissue removed from the nose in cases of retrobulbar neuritis, Herzog,[33] O. Beck,[5] J. Beck, White,[71] Oliver and Crowe[54] examined the mucous membrane of the ethmoid and found typical hyperplastic changes. Such findings are, of course, by no means significant of retrobulbar neuritis. Particularly O. Beck,[5] Oliver and Crowe [54] found a great amount of eosinophiles, in their cases pointing to allergy as a possible etiologic factor. As far as bone changes were concerned, Herzog[33] found definite destruction; J. Beck, definite apposition; while in the cases of O. Beck[5] the bone was normal. Inasmuch as all of these changes are observed in every case of hyperplastic ethmoiditis without retrobulbar neuritis, the thought that a particular form of ethmoiditis leads to retrobulbar neuritis must be negated.

Of greater importance are the cases of de Kleyn. The first case of de Kleyn and Gerlach[41] concerned a woman, age 48 years, who died of a pneumonia. Clinically, both nostrils were filled with pus, but on account of a marked ulcerative conjunctivitis, the eyegrounds could not be examined. At autopsy, a chronic, purulent pansinusitis was found, producing an ulcerative inflammation of the nasal mucous membrane. In the right sphenoid there was an ulcerative inflam-

mation of the mucous membrane penetrating into the marrow spaces of the bone, producing a marked destruction. A circumscribed portion of the sheaths of the optic nerve adjacent to the sphenoid sinus had disappeared and was replaced by newly formed connective tissue containing an infiltration and encapsulated cocci. Finally, there were small infiltrations in the connective tissue septa of the optic nerve. It was possible to follow in serial sections the infiltration along a venule which originated in the arachnoidal sheath of the optic nerve, perforated its dural sheath and continued toward the sphenoid sinus.

The second case of de Kleyn and Gerlach[42] concerned a female patient who had a large central scotoma for white and colors, and almost a normal peripheral visual field on the left side. Later, the patient acquired a definite choked disc on the right side, while on the left the choked disc was suggestive. At autopsy a fibroendothelioma of the dura was found in the left anterior fossa surrounding the optic nerve in the foramen opticum. Although on the left eye for a period of at least seven months there was a central scotoma, the optic nerve did not reveal any pathology.

The third case of de Kleyn[40] was a patient, age 51 years, who succumbed to a pneumonia following a phlegmon of the face. Antemortem, he had an enlargement of the blind spot for white and colors. At autopsy the mucous membrane of the sphenoid was observed to be very edematous. There were septic thrombi within the veins of the mucous membrane and within the marrow of the bone, extending into the cavernous sinus. However, the central retina vein was normal. The dural sheath of the optic nerve was slightly inflamed but the optic nerve was normal. In this case the first stage of a retrobulbar neuritis was found; *viz.*, an enlargement of the blind spot for white and colors. Pathologically, there was an osteomyelitis of the wall of the sphenoid, an acute inflammation of the mucous membrane with septic thrombi within the veins and a slight inflammation of the dural sheath of the optic nerve.

Redslob (quoted by Gifford[25]) saw an extension of a purulent sphenoiditis through the bone to the nerve in a child who died of a bronchopneumonia. Pickford (quoted by Gifford[25])

observed a perforation through the sella turcica allowing for an invasion of the nerve, with cocci demonstrable in its sheaths. Neither sphenoiditis nor optic neuritis were evident during life. Coffin[14] observed a child at autopsy with a thrombosis of the veins in the ethmoidal wall, orbit and nerve. The child clinically evidenced signs of an orbital phlegmon.

From only a part of the above-mentioned cases can conclusions be drawn as far as retrobulbar neuritis of rhinogenic origin is concerned, inasmuch as in these cases either a clinical examination of the eyes is lacking or there was a very marked purulent disease of the orbit and the walls of the sphenoid, none of which occur in ordinary types of retrobulbar neuritis. Therefore, for a possible solution to the point in question, only the Cases 2 and 3 of de Kleyn come into consideration. These cases show: *1.* That a retrobulbar neuritis can be existent for a period of several months without producing demonstrable changes within the optic nerve; and *2.* that there is such a disease as a rhinogenic retrobulbar neuritis which develops in a manner that an inflammation extends along the veins and the bony marrow from the mucous membrane of the sphenoid into the dural sheath of the optic nerve and, finally, into the nerve itself. Considering the anatomical and therapeutic facts previously mentioned, in addition to the microscopically examined case of de Kleyn, there can be scarcely any doubt that a rhinogenic retrobulbar neuritis *per se* actually exists.

IV. DIAGNOSIS OF RHINOGENIC RETROBULBAR NEURITIS.

Inasmuch as the writer does not feel fully competent to discuss the ophthalmological point of view, it should be recalled that many years ago Birch-Hirschfeld considered the central scotoma for colors, and later Van der Hoeve[35,36] considered the enlargement of the blind spot as the incipient stage of a retrobulbar neuritis. In the following years these two symptoms were sought by many investigators, with varying results. While, for instance, Markbreiter[50] found defects of the visual field in 70 per cent of cases with empyema of the sinuses, O. Beck[4] saw among 91 cases of empyema only six cases with a restricted field, and in only one instance among these evidenced the sign of Van der Hoeve. Therefore, it seems that neither the sign of Birch-Hirschfeld nor of Van

der Hoeve are very dependable. This was also pointed out by Cords.[17] But even if these signs were dependable, the ophthalmologist would only be in a position to make the diagnosis of retrobulbar neuritis, but not of a rhinogenic retrobulbar neuritis. Therefore, the question arises concerning the importance of nasal signs and symptoms for a positive diagnosis.

Unfortunately, there are no nasal findings which are characteristic of a retrobulbar neuritis. This lack of knowledge is due partially to the fact that it is customary for many rhinologists to publish spectacular cures and not actual cures of such cases. If one publishes a case of retrobulbar neuritis in whom, after an endonasal operation, a dramatic improvement of vision occurred, these cases do not prove very much from a scientific point of view. Of greater value are these cases in whom an endonasal operation produced an improvement of vision which, however, was lost immediately by an impairment of the findings in the nose or sinuses; for example, by a recurrent blockage of the sphenoid ostium. Most valuable, however, are the reports of patients in whom an endonasal operation was followed by a permanent improvement of vision and in whom symptoms of disseminated sclerosis did not appear during an observation of many months. Such cases must be actually considered as retrobulbar neuritis of rhinogenic origin. Inasmuch as there are few such cases on record, we can only enumerate the nasal findings which may be combined with what is termed retrobulbar neuritis of rhinogenic origin.

1. The Common Cold: The pathology of the common cold is not entirely clear. The general view is that it is due to an infection, allergy, or a combination of both. The common cold, which occasionally leads to a retrobulbar neuritis, clinically makes the impression of an infection rather than of allergic origin, inasmuch as it is frequently combined with fever and general malaise. Particularly, this form of retrobulbar neuritis is often considered as rhinogenic in origin because Oliver and Crowe,[54] Herzog[34] and others have emphasized that acute infections of the nasal mucous membrane may readily reach the deeper layer of the mucous membrane. Below a mucous membrane which shows but moderate pathologic changes there may be severe inflammation of the bone marrow. Furthermore, it is possible that the slight changes

on the surface such as swelling and the loosening of the mucous membrane may subside a few days after the acute attack, while occasionally the inflammation in the marrow spaces persists. This has been emphasized by the findings of Stenger,[63] O. Beck,[5] J. Beck and others.

It is necessary, however, to evaluate these findings with some degree of critical analysis. In the first place, inflammation and infiltration with eosinophiles of the mucous membrane of the nose and of the ethmoid are found so frequently that it is difficult to decide where the physiologic change ends and where the pathologic alteration begins. As a matter of fact, some pathologists speak of a "physiological inflammation" of the mucous membrane of the upper respiratory tract. But even if such findings are considered as pathological, they only prove that a retrobulbar neuritis may occur in acute inflammation of the nasal mucous membrane. However, they do not prove that retrobulbar neuritis is actually due to the inflammation. On the contrary, from a theoretical point of view, it is possible to conclude that the infection or allergy which has produced the common cold, simultaneously has also produced the retrobulbar neuritis. This concept also holds true when one considers the fact that in many of these instances the neuritis appears after the common cold has subsided. Furthermore, the fact that in these cases the treatment of the nose, for example with adrenalin, frequently improves vision, only proves that an inflammation within the optic canal might be improved by a treatment of the nose. It does not, however, prove that an acute inflammation of the optic canal was due to the inflammation of the nose. Unfortunately, we must conclude that in these cases, theory against theory is promulgated and an actually well founded knowledge is lacking.

2. Manifest Empyema and Hyperplastic Inflammation of the Sphenoid and Posterior Ethmoids: Many investigators (White,[71] Stenger,[63] Herzog,[34] Beck and Pillat[6] and others) have emphasized that these findings are rare in retrobulbar neuritis. In 435 cases of chronic nasal suppurations, Herzog[34] found only six cases (about 1.4 per cent), and in 65 cases of acute nasal suppuration, seven cases (about 10.7 per cent) of retrobulbar neuritis. Bachstez[2] observed among eight cases of rhinogenic retrobulbar neuritis, four patients with a

manifest empyema of the ethmoid. Three of these patients were cured by opening the ethmoids, one by treatment with adrenalin and mirion. The cures in these cases lasted from six to 10 years. In a fifth case of Bachstez[2] there was a hyperplastic inflammation of the ethmoid. Cure was obtained by opening of the ethmoid, and its cure persisted for seven years. Probably in the case of Elles[21] there was also a hyperplastic inflammation of the sphenoid. The patient was cured by the opening of the sphenoid, and remained well for five years. Among the cases of Oliver and Crowe[54] there were three cases with hyperplastic inflammation in the posterior sinuses which were cured by operation. These patients remained symptom-free for a period of over one to five years. In two other patients there was a suppuration in the posterior sinuses; both were subjected to surgical intervention. One patient had normal vision for 18 months, and in the other the visual field rapidly increased after operation and then contracted somewhat. Nevertheless, the patient had useful vision two years after the operation. Stenger[63] emphasizes that even in these rare cases in whom a chronic inflammation of the paranasal sinuses was found, the retrobulbar neuritis was usually preceded by an acute exacerbation of the inflammation. Consequently, the neuritis in such instances is not due to a slowly progressive inflammation but to an acute exacerbation.

3. Latent Inflammation of the Ethmoid and the Sphenoid: These cases are particularly emphasized by ophthalmologists. Van der Hoeve[37] states that the rhinologist cannot say with absolute certainty that a person has no sinus infection. Meller and others share this opinion. There is no question that this point of view was perfectly correct some years ago. However, in the meantime, the diagnostic ability of the rhinologist has advanced so that the number of the latent diseases of the paranasal sinuses has considerably decreased. Although such an outstanding Roentgenologist as E. G. Mayer[51] claimed that in cases of retrobulbar neuritis, the paranasal sinuses are usually found to be normal, de Kleyn and Stenvers[43] had the opposite experience with the oblique exposure after Rhese; Shambough,[61] with the filling of the sinuses with lipiodol; Cone, Moore and Dean,[16] with body section radiography of the sinuses. Therefore, undoubtedly many cases of

inflammation of the sphenoid which were considered as latent some years ago are now being uncovered by the better diagnostic means at our disposal.

Even if one insists on the frequency of latent inflammation of the sinuses, it is difficult to understand why a chronic inflammation of the sinuses seldom produces a retrobulbar neuritis if it is manifest, while it frequently produces disturbances of vision if it runs a latent course. To meet this contradiction it was assumed that the latent chronic inflammation of the sinuses serves as a focal source of infection and thus resulting in a retrobulbar neuritis. White,[73] in particular, advocates this principle and attributes greater importance to the teeth and the tonsils than to the infected ethmoid, insofar as the etiology of retrobulbar neuritis is concerned. The main reason emphasized by White[73] to support his concept is the effect of the treatment upon the condition. This reason generally also satisfies the clinician, and it must satisfy him, inasmuch as the research work has not created facts as yet to confirm or to refute this opinion. Nevertheless, it should be borne in mind that therapeutic results do not always permit conclusions insofar as the etiology is concerned. The following case may prove the correctness of this statement:

A man, age 51 years, became ill with a serous meningitis, which, on account of a misinterpreted X-ray picture of the sinuses, was believed to be due to a suppuration of the right frontal sinus. By an external approach, the right frontal sinus and the right ethmoid were opened. However, the patient had no frontal sinus and, thus, after the removal of a part of the frontal squama, the normal dura was exposed. In the ethmoid there was a definitely hyperplastic mucous membrane, which was removed. After the operation, the patient made an uneventful recovery. X-ray examination of the teeth, however, which was carried out later, revealed 11 granulomas. If in this case extraction had been made of the 11 teeth, and subsequent recovery ensued, undoubtedly the cure would have been attributed to the elimination of the dental foci. However, in this instance the meningitis was cured without tooth extraction. Whether this cure was due to the exposure of the dura or to the opening of the ethmoid, or to some other factor, is uncertain.

4. Disturbances in the Ventilation of the Nose: These cases are most questionable, inasmuch as they do not present pathology which can be diagnosed by the usual clinical methods. In a number of these instances, certainly, a latent chronic inflammation of the ethmoid is present; however, these cases are not as frequent as many ophthalmologists believe. In other cases, anomalies of the anatomy of the nose are held to be responsible. White[71] found an enlargement of the turbinal tissue adjacent to the sphenoidal ostium in most of his patients. He believes that the size and position of the middle and superior turbinates probably explain the etiology in many instances. Poor ventilation and faulty drainage are all important predisposing factors. Stenger[63] and Skillern[62] also emphasize the importance of a disturbed nasal ventilation in the etiology of retrobulbar neuritis. According to Stenger, these disturbances are due to the following findings: *a.* The middle turbinate is displaced backward. It is small and hardly visible as the result of an enlarged inferior turbinate or a deviated septum. Employing posterior rhinoscopy, one finds a narrow and low choana on the diseased side. The entire ethmoid seems to be displaced backward and downward. *b.* Pneumatic cells within the middle turbinate; and *c.* abnormal size of the ethmoid bulla. It is more than questionable whether or not such findings can be actually considered as etiological factors of retrobulbar neuritis. The findings mention under *a.* are so often without any influence upon the optic nerve that they scarcely can be held responsible for producing a retrobulbar neuritis. As far as the other findings are concerned, Beck and Pillat[6] have examined such noses but were unable to find even the slightest changes in the visual fields.

Summarizing, it is clear that there is no nasal finding characteristic of retrobulbar neuritis. Even in cases of common cold it is difficult to say whether a retrobulbar neuritis is the result or an accompanying symptom of the acute nasal inflammation.

V. DIFFERENTIAL DIAGNOSIS.

Rea[57] enumerates the following diseases which (except the paranasal sinuses) may produce a retrobulbar neuritis: *1.* Neuromyelitis optica; *2.* syphilis; *3.* disseminated sclero-

sis; *4.* Leber's disease; *5.* tobacco amblyopia; *6.* vascular disease; and *7.* hysteria. Bachstez[2] adds to this list the stationary scotomatous atrophy of the optic nerve of Jensen, tumors at the base of the frontal lobe of the brain, injuries of the chiasm, general sepsis and, particularly, tuberculosis. Tuberculosis was also emphasized by Friedinger[23] and by Loewenstein,[19] who found Koch's bacilli in the blood stream of patients suffering from retrobulbar neuritis. The latter finding was refuted by Kolle and Kuester.[14] Among all of these conditions the most important are: *a.* toxic amblyopia; and *b.* disseminated sclerosis.

a. Toxic Amblyopia: Considering the history of the patient, it is generally not difficult to make a diagnosis. Nevertheless, one might meet with difficulties when in such an instance definite nasal pathology is found. This happens quite frequently. Forschner and Sommer[22] found among 12 cases of toxic amblyopia eight instances of hyperplastic inflammation in the paranasal sinuses. These authors believe that the permanent hyperemia and infiltration, as well as the chronic edema of the nasal mucous membrane, render the optic nerve more susceptible to the poison. Naturally this is an hypothesis. However, the following case proves the difficulties one may expect in such cases. A man, age 25 years, had an external frontoethmoid operation in April, 1937, for an obstinate nasal polyposis complicated by a chronic bronchitis. The vision was entirely normal. In July, 1940, the right side of his nose was reoperated for an acute infection of the cavity. On Oct. 22, 1940, he returned, complaining of impaired vision which followed a common cold a few days before. The eye examination on Sept. 11, 1940, did not reveal any pathology. However, on Oct. 22, 1940, a primary atrophy of both optic nerves was found. The neurologic examination was negative, and in the nose itself there were neither polyps nor pus. Since it was assumed that the disease of the optic nerve had originated in the nose, the left side was reoperated, but only scar tissue was found. After this operation the patient was just by chance discovered in the act of drinking denatured alcohol, and confessed to the fact that he drank a great deal, occasionally even wood alcohol.

This is undoubtedly a case of toxic amblyopia combined with considerable pathology in the paranasal sinuses. As a

matter of fact, the proper treatment with vitamins and abstinence from alcohol definitely improved the vision of the patient.

In order to find out whether the retrobulbar neuritis is of toxic or rhinogenic origin, Ruttin[58] recommended the examination of the ears in such cases. He found, among 25 cases of acute retrobulbar neuritis, 20 instances where there was pathology of the internal ear. He concludes that in the presence of an affection of the optic nerve, as well as of the acustic nerve, the toxic origin of the disease is more likely than the rhinogenic origin. As a matter of fact, Forschner and Sommer[22] observed 12 cases of toxic amblyopia in eight instances of which a disease of the internal ear was present. The same finding was met with in one case of arteriosclerosis, in one of lead poisoning, and in one of syphilis. Of particular interest is the fact that in six instances of rhinogenic retrobulbar neuritis the ears were normal.

b. Disseminated Sclerosis: As mentioned above, there is no doubt that disseminated sclerosis produces retrobulbar neuritis more frequently than do infected paranasal sinuses, although the importance of the former should not be exaggerated. Inasmuch as the diagnosis of an incipient disseminated sclerosis is very difficult, it is obvious that in such a case one neurologist may make the diagnosis very emphatic while another refutes the diagnosis. This is the reason why the frequency of disseminated sclerosis in the etiology of retrobulbar neuritis varies between 7 and 73.1 per cent. It is, of course, not possible for the rhinologist to make a definite diagnosis in such a case when the neurologist himself is uncertain. Nevertheless, there are certain findings which allow a tentative diagnosis of a disseminated sclerosis, providing that the general examination of the patient does not reveal any findings indicating a toxic source due to lead, syphilis, tuberculosis, etc. Such findings are the following: *a.* Finding a normal nose, even by means of the modern methods of examinations. *b.* Type of the disturbance of vision: Hensen[21] emphasized that, in general, the central scotoma in disseminated sclerosis is very fugitive, provided that the affection of the optic nerve appears at the onset of the disease. As a rule, the disturbance of vision is cured in three to four weeks. Of course, there are occasionally also more obsti-

nate and even permanent disturbances of the vision. Therefore, the duration of the central scotoma allows certain conclusions, insofar as the etiology is concerned. According to Hensen,[31] disseminated sclerosis never produces a scotoma which lasts longer than three months, unless there are also other neurological signs and symptoms of the disease. In other words, disseminated sclerosis produces either — that is the rule — a central scotoma which disappears in a relatively short period of time, or there is a more severe affection of the optic nerve, the central scotoma persisting for a longer period of time does not show any tendency toward recovery. However, in the latter instance, the optic nerve is never the sole sign or symptom of the disease. These findings are very valuable, inasmuch as they were described under the supervision of Wilbrand, who possessed an extremely wide experience in this field. Also, Lagrange et Marquézy[46] never observed a complete atrophy of the optic nerve in disseminated sclerosis, but rather a temporal pallor of the disc. *c.* I[12] have emphasized that in cases of retrobulbar neuritis due to disseminated sclerosis, there are frequently also labyrinthine symptoms such as dizziness and nystagmus. Therefore, given a case of retrobulbar neuritis in whom all clinical findings, including the neurological and otological findings are negative and labyrinthine dizziness and/or nystagmus are observed, a tentative, although not the definite diagnosis of disseminated sclerosis can be made. Such an instance is illustrated by a case described by Bollack.[9] In his patient, a retrobulbar neuritis preceded a disseminated sclerosis by three years. However, at the time when the patient suffered from the retrobulbar neuritis, he also complained of *quelques étourdissements et vertige, pas de bourdonnements d'oreille.* Unfortunately, Bollack[9] does not give an exact description of the dizziness. However, it seems likely that the patient suffered from a labyrinthine form of dizziness. The syndrome of retrobulbar neuritis and labyrinthine symptoms in incipient disseminated sclerosis can be understood when one recalls that, according to the statistics of Brain[11] concerning disseminated sclerosis, the frequency of pallor of the disc is approximately 44.3 per cent, that of nystagmus approximately 67.6 per cent, and of dizziness approximately 32.88 per cent. It is, of course, questionable whether or not in all of these cases, actually labyrinthine dizziness and nystagmus

were considered, inasmuch as unfortunately even at the present time, neurologists frequently do not appreciate the characteristics of labyrinthine dizziness and labyrinthine nystagmus. Nevertheless, clinical experience proves that in the majority of these cases one actually is concerned with labyrinthine dizziness and nystagmus. This state of affairs explains the joint appearance of retrobulbar neuritis and labyrinthine symptoms in incipient disseminated sclerosis.

Naturally, even in following these clues and in using the available modern methods of rhinology, pitfalls cannot be avoided, although they should occur less frequently than in the past.

VI. TREATMENT.

In this review, only the local treatment of the nose in retrobulbar neuritis is to be discussed. Such management is either medical (conservative) or surgical.

Conservative Measures: The lavage of the nose, according to the technique of Proetz, was used by Shambaugh[51] and others with success. Benedict[8] applies a 2 per cent solution of iodine, or places in the nose, two or three times a day, for three hours, cotton saturated with a mild solution of silver proteinate. Herzog[34] and Herrenschwand[32] recommend a similar pack treatment, using adrenalin. They place a strip of gauze soaked with suprarenin into the middle meatus twice daily for two hours. The treatment is carried out from eight to 14 days.

Surgical Procedures: A distinction must be made between the more conservative and the radical methods. Conservative measures consist of the displacement of the middle turbinate and the resection of the septum. To the radical methods belong the resection of the middle turbinates, the opening of the bulla, the opening of the ethmoid and enlarging the opening of the sphenoid. De Kleyn[39] and Skillern[62] believe that these procedures influence the process less by procuring a good drainage than by producing a normal circulation of blood in the posterior part of the nose and in the optic canal. Benedict[8] believes that the cures obtained by surgery may be due to the cocaine-epinephrine analgesia which produces an ischemia and then a congestion in the vessels of the optic

nerve, and, then, to the absorption of blood following the operative trauma which leads to a rise in temperature. These two factors apparently produce a response analogous to that which is induced by injections of foreign proteins.

Indications: A great number of contributions deal with this problem, although a number of them merely reiterate that which has been common knowledge for many years. First, as far as the conservative treatment is concerned, it can be employed in every case, even in cases of retrobulbar neuritis resulting from a common cold or in cases of evident disseminated sclerosis. The only risk one assumes is that the treatment is superfluous. I do not believe that there is a great difference between the various methods of treatment, inasmuch as probably each type of medical procedure which affects the circulation of blood in the posterior part of the nose is efficacious in selected cases. I personally employed the treatment with adrenalin packs for many years. The only disadvantage of this form of treatment is that it takes a long time.

As far as the more conservative methods of surgery are concerned, practically the same holds true as that which was said concerning the purely conservative medical treatment, except, of course, that surgery should be avoided in patients suffering from an acute cold. The problem begins with the more radical types of operative procedures. However, there is but little that can be discussed concerning such cases which require radical operative intervention, from the rhinological point of view. The fact that the patient has a retrobulbar neuritis or a disseminated sclerosis does not influence the indication for an operation upon the paranasal sinuses, providing that they are definitely affected. It is quite different under those conditions which we have to deal with when the nasal findings are questionable or negative. These are actually the cases about which much of the discussion centers.

It has already been mentioned above that by means of modern methods of rhinologic study, the number of questionable or negative cases in retrobulbar neuritis has considerably decreased. Nevertheless, there are still a number of cases in whom even the modern methods fail to show pathology in the nose. In these cases the rhinologists follow a different con-

cept. First, there are rhinologists who demand an immediate opening even of normal parasanal sinuses if a definite etiology of the retrobulbar neuritis cannot be found (v. Wildenberg,[74] Gallaher,[24] Skillern,[62] Escat, Onodi,[55] van der Hoeve,[37] de Kleyn,[39] Sourdille and many others). The reason for this concept is: *a.* because an atrophy of the optic nerve may soon result, according to Gallaher,[24] in 15 to 20 days; and *b.* because a clinically normal nasal mucous membrane may disguise a severe inflammation in the marrow spaces of the bone, thereby injuring the optic nerve (Meller and Hirsch,[52], Oliver and Crowe[54] and Herzog[34]).

To the above-mentioned reasons I wish to add a third one, which does not originate from a purely scientific but rather from a practical point of view. One must bear in mind that there is no recognized adequate treatment of disseminated sclerosis. Brain[11] states "the multiplication of remedies is eloquence of their inefficacy. The cure of disseminated sclerosis awaits an increase in our knowledge of the causal organism, of the nature of immunity and of the factors upon which depend the remarkable variations in the course of the disease." Therefore, being in an experimental stage, the treatment of disseminated sclerosis could also include the opening of the paranasal sinuses, among other experimental applications.

Despite such reasoning, I do not agree with this point of view, inasmuch as the proposed operations are not to be considered entirely without danger. First, one has to bear in mind that we are concerned with mutilating types of operations, any of which may occasionally be followed by severe complications, such as emphasized by Cushing,[18] Bordley,[10] Davids[20] and others. Symptoms of slight or severe meningitis occur after these operations even more frequently than is recorded. Schnaudigel (quoted by Cords[17]) mentions a case in whom the optic nerve was injured in curetting the posterior ethmoid. Bordley[10] reports a similar case. I have observed the following case: T. W., age 57 years, suffered from a common cold a few years ago, probably with affection of the paranasal sinuses. In April, 1937, she noticed impaired vision in the left eye. The superior portion of the visual field was foggy. A diagnosis of retrobulbar neuritis was made by Dr. Pressburger. Treatment with adrenalin packs was carried out. This was followed by a further impairment

of vision. On April 15, 1937, an amaurosis of the left eye, with an absence of the pupillary reaction to light was observed. Therefore, an opening of the sinuses was indicated. Nasal examination revealed a slight deviation of the septum to the right; the anterior end of the left middle turbinate was enlarged, but the middle meatus was wide. There was no evidence of pus or hyperplasia. On April 16 the left ethmoid was opened after the technique of Halle, and the anterior wall of the sphenoid sinus was removed. The mucous membrane of the anterior part of the ethmoid was slightly edematous but the mucosa of the sphenoid was normal. In the anterior end of the middle turbinate was a pneumatic cell. Microscopic examination revealed a slight inflammation of the mucous membrane of the ethmoid. On April 20, a slight pupillary reaction was noticed, but the temperature rose to 104° F. Yatren-casein injections were administered, and on April 23 the temperature fell to 98.6° F., and the patient could see the light before the left eye. On April 28 the patient noted paresthesias in the lower extremities, followed by a quadruplegia. The diagnosis of myelitis was made. On July 1 the patient could see fingers before the left eye, but there was a rapid decrease of her general condition and a large decubital area developed. The motility of the arms was free but that of the legs was markedly diminished. The temperature varied between 98.6° and 100.5° F. She was treated with Betaxin, Urosept and Prontosil, and was dismissed on August, 1937, with an atrophy of both discs, and slightly improved vision. Shortly after her dismissal, the patient died. No autopsy was performed.

In this case the operation on the paranasal sinuses, which were practically normal, was followed by a neuromyelitis optica, which many neurologists consider as a special form of disseminated sclerosis. Although there is not the slightest evidence that the operation produced the neuromyelitis optica, unquestionably surgery did not stop the course of the condition, but perhaps accelerated the progress of the disease.

According to White,[72] the indication to open the paranasal sinuses depends upon the size of the optic canal as shown on the X-ray examination. An optic canal of 4 mm. or less, in a case of severe optic nerve involvement, indicates the necessity for immediate ventilation of the posterior sinuses to

prevent permanent atrophy. It seems that the point of view emphasized by White[72] does not meet with general acceptance.

Despite these arguments, normal sinuses are still opened in these cases too frequently. Lillie,[47] in more than 500 cases observed, found that in more than 60 per cent of the cases of retrobulbar neuritis or optic neuritis, a paranasal sinus operation had been done elsewhere some time during the course of the illness.

I believe that in accordance with the present state of our knowledge it is necessary to combine the conservative and surgical management in these cases. This was emphasized by Grosz,[27] White,[73] Bachstez,[2] Herzog,[34] Janssen,[38] Whelan[69] and many others. First, a trial should be given to the conservative treatment, which, according to Grosz[27] and White,[73] can be continued for one to two weeks. If this treatment meets with no success, the surgical intervention comes into consideration.

Against this concept, it is usually emphasized that during such a waiting a retrobulbar neuritis might progress into a permanent atrophy of the optic nerve. However, the experience proves that in patients with a retrobulbar neuritis, in whom the affection of the optic nerve progresses rapidly, the opening of the paranasal sinuses fails to stop the degenerative process unless there is definite pathology in the paranasal sinuses.

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307 North Michigan Boulevard.

MILITARY SYMPOSIUM.

WHAT WAS WRONG WITH OTOLARYNGOLOGY IN THE LAST WAR.*

DR. HARRIS P. MOSHER, Marblehead, Mass.

Some months ago at about the time when medical preparedness began to be taken seriously, and the A. M. A. sent out its blanks and the National Research Committee appointed its committees, I carried out an idea which I had had long in mind; namely, to assemble criticisms of the Otolaryngological Service in the last war. Accordingly, I sent letters to a number of men and received answers from them all; that is, 13. They were written thoughtfully and with a desire to help.

Naturally, I knew that certain things were wrong with Otolaryngology in the last war because I shared the responsibility for some of them. I wanted, however, to close the books, as it were, hoping that if these things were put on paper, those who carry on during the present mobilization, and into the next war if it comes, would be helped to do a better job than was done last time.

By way of introduction I shall start off with the only fully pleasant letter which I have at hand. You will notice that it was written by me to Dr. Forbes, of New York, in answer to one from him written in the first glow of reaching home from overseas; that is, some 20 years ago, and some might say, written without due consideration on his part, but he adds a note written this year when he sent the letter to me, saying that he still feels the same, bless him!

(In reply to S. G. O.)

WAR DEPARTMENT
Office of the Surgeon-General
Washington

Feb. 3, 1919.

Dr. H. H. Forbes,
40 East 41st Street,
New York City, N. Y.

Dear Dr. Forbes:

I have just received your letter of Jan. 29, written on board the U.S.S. Plattsburg. Thank you for letting me know that you are back. It pleases

*Read at the Seventy-fourth Annual Meeting of the American Otological Society, Inc., Atlantic City, May 27, 1941.

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me very much that you can say that you have had a wonderful experience and are grateful for the opportunity. This is not the universal feeling of men coming back. In fact, more than one has expressed the opinion that it was not in any way worth while. Sincerely,

HARRIS P. MOSHER,
Lieut.-Colonel, Medical Corps.

Note: Jan. 21, 1941.

Rather pleasing letter. I feel the same today and sincerely wish for a part in the big effort now being made. H. H. FORBES, M.D.

The order of what I have to say today is to give the conclusions drawn from the 13 letters, and then to discuss certain of these. I am tempted to read extracts from the letters, giving the names of the authors, for names, as newspaper men say, have great news value. On second thought, this seems to be going a little too far, and I shall omit the abstracts. They might get someone into trouble. As for myself, it does not matter.

SUMMARY OF CRITICISMS.

Too many recruits with chronic running ears were missed by the Medical Draft Boards and passed into the service.

No recruit with a chronic running ear should be passed except for limited or special service. Such recruits are notorious pension seekers.

The special equipment for Otolaryngology often did not reach the overseas hospitals, and much that came through was poor.

In Otolaryngology, too many medical officers used the Base Hospitals chiefly in order to gain experience in operating.

Too many radical mastoid operations were performed.

Superior rank in a medical officer did not always mean superior training and mature judgment. Promotions overseas at times smelled of politics or personal favoritism.

The inborn jealousy of some of the regular Army medical officers of the civilian medical officer too often came to the surface.

Too often the specialist stood on what he considered his rights and was not a good sport when his hospital was in a jam.

Army medical schools for training specialists were wasted effort. Refresher courses for real but not for self-styled specialists might have been worth while.

Most operations for rehabilitation should have been delayed until after the war, except for some plastic surgery of the face.

In the present army medical setup, a roving consultant should be appointed for specified areas. He should make regular rounds, determine what type of operations should be done, and make recommendations to straighten out obvious injustices in rank. Naturally, he should be a man of mature judgment and wide operative experience, and well known.

In the Surgeon-General's office and in Base Hospitals, Ophthalmology and Otolaryngology should be independent of Surgery. In the field, surgery should command.

A Head Hospital should be staffed by an ophthalmologist, an otolaryngologist, a neurosurgeon who is a good neurologist, and a plastic surgeon familiar with dental technique.

The treatment and prevention of flu and respiratory diseases in general were major unsolved problems of the last war and rank the same in this one. Mass immunity by the use of appropriate sera and chemotherapy offer hope that progress in the prevention and treatment of respiratory infections in cantonments and base hospitals may soon be made, and epidemics prevented.

DISCUSSION OF CRITICISMS.

The chief criticism of the Otolaryngological service was that the special equipment failed to reach hospitals overseas, or if it did reach the other side it was stalled in supply depots. I know that this is true, because at the end of the war the thrifty French (not to use a stronger word) profited by a large amount of unopened American equipment.

Too many Otolaryngologists used the Base Hospitals in order to gain surgical experience. Their rank gave them the opportunity to operate on their own responsibility, but their previous training and experience did not fit them for it. The result was that far too many radical mastoid operations were

done. A general order from headquarters finally called a halt to this.

This brings up the question of the roving consultant. Such a plan was carried out overseas but not in this country. A few inspection trips were made by the personnel of the Surgeon-General's Office but no medical officers were assigned to act as traveling consultants. In the present medical setup in this country, there is work for such consultants even now, and if there ever should be an overseas expedition again, such consultants should function from the very first. It should be the duty of this board of consultants to make regular rounds in specific areas. They should decide what type of operations are to be done and make recommendations to straighten out obvious injustices in rank. The consultants should be men of proven tact, men outstanding in the profession and well known. It goes without saying that they should be men of mature judgment and wide operative experience. This sounds a bit like setting up a Gestapo as they have in the German Army, but the tact of the consultant should prevent the position from sinking to this low level.

The chronic running ear was a problem in the last war and holds over to this one. Men with this disability got by the draft examinations in large numbers, only to swell the pension lists after the war was over. Something of the same kind I feel is happening now. In the Canadian Medical Corps they make a strong point of detecting these men. Such men, if admitted to the service at all, should be given special duties for which they are fitted, and the degree of their hearing disability carefully recorded on their admission blank.

Recently I received a letter from a London friend who is in the English Medical Corps serving in the East. He was in Athens at the time and before the German occupation. Speaking of things medical, he said that he never realized how much otorrhea there was in the Army.

The basic plan of war is destruction. Even in the preparation for war this fact is evident. An instance of this is the loss of hearing due to gun fire. Men who set up or inspect airplane engines also are subject to extreme noise. The same is true of aviators unless they protect their ears. It has been noticed that certain susceptible aviators have a progressive

loss of hearing, a loss which may make them deaf to the radio beam. As the question has been raised how often this happens, those who have the medical care of aviators should settle the question at once by regular and periodic examinations. There is an ugly suspicion that failure to hear the radio beam was responsible for some of the recent commercial airplane crashes.

Being called upon to do other than strictly specialists' work was an outstanding grievance among the poorer men. I say poorer advisedly, because those who fully sensed what war meant were continually breaking the bounds of their specialty, and got a lot of satisfaction in helping out where help was needed. In war, a man should be willing to turn his hand to anything if the occasion calls for it. Not to do so shows that he is a poor sport.

Another cause of complaint which was often heard, especially during the waiting period in this country before orders to go overseas came through, was that there was little or no work for the specialist to do. This was inevitable and is bound to occur again. Philosophy is the only help under such circumstances, plus keeping in mind that those serve "who only stand and wait."

Such spare time could have been spent profitably studying hospital organization or learning paper work. Then there was always the broad subject of sanitation. Be bigger than your specialty is not bad advice in the Army or out of it.

Rank caused many heartaches and many complaints. It is easy for a man in a specialty to evaluate the training and ability of another man doing the same line of work. When, therefore, a man of fuller experience is outranked on the same service by a man of lesser training, the atmosphere at once becomes overcharged. The rank which a medical officer holds cannot be said to be entirely a matter of pride on his part. It determines in most cases the amount of money the family back home have to live on. Once in the Army, persistent gossip has it that rank at times is settled by the commanding officer on personal considerations. While human nature remains as it is, this may happen occasionally.

You cannot make a competent, safe specialist by Army training schools. A splendid attempt was made at Ogle-

thorpe. The effort was most conscientious but the results did not pay for the labor expended. DeSchweinitz, on an inspecting trip, once asked a self-styled specialist whom he met at Oglethorpe, "By what method of reasoning did you arrive at the conclusion that you were a specialist in eye?"

In time of war, the regular army serves as a skeleton unit about which expanding civilian units are built. The army is helpless for any big job without outside help. Organization is its strong point and paper work its god. As it is really helpless alone, a certain humility would seem to be in order, but, unfortunately, is not always apparent. What I am struggling up to is this — there is a certain amount of inborn jealousy on the part of some of the regular army medical officers of the civilian medical officer. This at times comes to the surface, rising even to insolence. I have heard more than one complaint on this score. By some, this attitude has been assigned to an inferiority complex, but I am not prepared to agree fully with this opinion. The fact remains that this thing exists. For example, take the happening which occurred to one of the members of this Society who is now listening to me and wondering how far I am going. When he reported to his superior medical officer in the last war, he was asked if he did any special type of medical work. He answered that he was an Otolaryngologist, and received this blast in the tone of the old-fashioned drill sergeant and accompanied by an oath — "I'll show you specialists where you belong." Authority, not breeding, was responsible for such a remark. At one of the present cantonments recently, some of the recruits objected to the browbeating of the drill sergeant. News of this got into the press and word soon came down from above to stop it. I am not suggesting kid gloves in handling men. I have had something of an experience along this line and have never been accused of owning a pair. Those who have not experienced this thing will say that I am making much out of little. Those, however, who have felt its sting think very differently.

Having criticized some of the regular Army medical officers, it is only fair to record a criticism justly raised against some of the civilian consultants. For example, it happened occasionally that a consultant when visiting — really inspecting — his special department of a hospital, stalked at once to the department in question without first reporting to the com-

manding officer. This was extremely bad manners. How would you feel were you the commanding officer? Such a happening is a neglect of a natural, elementary and fundamental Army courtesy.

Modern war is a war of movement. Battles are now often won in a few hours or days. The wounded hinder movement and must be gotten to the rear as soon as possible. Even a base hospital may not be a stable affair. I don't think that an American could drive a tank over his own wounded, as rumor has it the Germans have done. Nevertheless, the wounded can be given hardly more than first aid at the front. A motorized hospital unit might save some of the desperate cases as they are carried to the rear.

A recent letter from Dr. John M. Converse, who is serving with the American Hospital in Britain in the Plastic Service, and who has acted as observer for the Committee on Otolaryngology in this country, said that a mobile Head Unit would be of great use in England at the present time.

The deadly flu epidemics, both in this country and overseas, were the most dramatic happenings of the war. It is not a pleasant thought, but it is true that man's most dangerous enemy is man. Herd, or assemble if you like a more polite word, a large number of nonimmune or partly immune men together as occurs of necessity in cantonments, and there is sure to be a carrier of infection among them; if not, a visitor brings it in. As a result, infection starts on a large scale and increases in virulence as it jumps from one human test tube to the next. Respiratory infections were the great problem of the last war and remain a major problem for this one.

There is hope in the preventive use of vaccines, and the sulfanilamide group offer a greater hope. It is a striking fact that after Dunkirk there was practically no tetanus in the English troops, due to the routine use of tetanus toxine during the training of the men. Naturally, our medical officers, having this in mind, proposed using it for our drafted men. The public immediately began to shout "experimentation" and "human guinea pigs," and for the moment, at least, the matter has, I understand, been dropped. A medical Army officer has to be a bit of a politician. More could be said on that subject if this were the place.

527 Front Street.

MILITARY SYMPOSIUM.

HEARING STANDARDS FOR ACCEPTANCE, DISABILITY RATING AND DISCHARGE IN THE MILITARY SERVICES AND IN INDUSTRY.*

DR. EDMUND PRINCE FOWLER, New York.

The basic factors necessary for establishing a dependable method for measuring percentage losses in hearing capacity from monaural and binaural deafness are herein set forth. These factors are important for estimating hearing losses in the personnel of the military services as well as in civil life and industry. Standards are set up upon which to base opinions as to the percentage of disability suffered by a normally hearing person, or one who in the past has had a partial deafness in one or both ears.

A table is constructed for estimating ***the percentage of loss of capacity to hear speech.*** *By changing the weighting it may be used in other categories of hearing function.*

The present hearing standards for the military services serve as practical "rule of thumb" tests for determining whether the hearing is or is not sufficient to hear speech or other sounds at specified distances, but it is apparent that more precise and dependable standards are desirable.

The hearing standards in the navy for enlistment or commissions are: for the whispered voice 15/15ths in each ear, and during general service the hearing must not fall below 7/15ths (binaural) for conversational voice; in the aviation service not below 7/15ths (binaural) for whispered voice.† For the personnel already in the service these standards are not so rigidly adhered to, but the hearing of all officers is examined annually, and on promotion; of all enlisted men on transfer, on promotion and on re-enlistment.

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*Read at the Seventy-fourth Annual Meeting of the American Otological Society, Inc., Atlantic City, May 27, 1941.

†The numerator indicates the distance the sound is heard, and the denominator indicates the distance the normal ear hears the same sound under the noise condition existing in the examining room.

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In the regular army the standard is 20/20ths in each ear.*

In the National Guard and reserves, 20/20ths in one ear and not less than 15/20ths in the other.

For Flying Cadets, Class I, 20/20ths in each ear, Classes II and III at least 8/20ths in each ear.

By audiometer, Class I, an average hearing loss not more than 15 per cent (obtained by multiplying the averaged readings at 512, 1,024 and 2,048 by 0.8). Classes II and III, no standards are set but qualifications are based on the whispered and the soft conversational voice tests.

For selective service low conversational voice is employed (not a whisper). (In case of doubt use a watch.)

When indicated, in addition to the above the audiometer is used according to the instructions furnished by the manufacturer.

Class 1-A: a. Normal hearing; *b.* 10/20ths or better in each ear.*

Class 1-B: a. Less than 10/20ths but at least 5/20ths in one ear. The opposite ear may be totally deaf.

Class 4: Hearing less than the minimum prescribed under 1-B.

In addition to these tests in the navy, the watch and acumeter or coin-click are sometimes used, but the regulations say, "the voice is a more reliable method of determining the acuteness of hearing than the watch test, as it allows for variations in hearing with the modifications produced by changes in pitch and tone, and the voice can be raised if there are noises in the vicinity of the examining room."

Whereas some of these tests have been found useful in the military services they are undependable, in fact useless for estimating percentage of disability within or without the services, or in industry, because with the exception of the

*The numerator indicates the distance the sound is heard, and the denominator indicates the distance the normal ear hears the same sound under the noise condition existing in the examining room.

audiometer all are basically unsound and misleading. These tests (still in use) are the reason for the dissatisfaction and controversy as to the amount of compensation due to veterans from loss of hearing in the last war. Except for checking purposes, all except the audiometer tests may be discarded with advantage if precise standards for the percentage of hearing capacity are substituted. If this is done, Uncle Sam and Uncle Sam's children will be saved much annoyance and injustice.

The audiometer tests as now generally used also are misleading because the decibel losses are averaged, or worse, are transposed into what is called "a per cent loss." A percentage loss of what? Certainly not the capacity to hear speech, because, as everyone knows, many people suffer far more (or less) disability than the figures so obtained indicate, hence the cry for tests by actual speech, which is what the soldier and civilian are mainly interested in.

I grant that the ability to hear speech is usually the most important function in hearing and that a man's disability from deafness should usually be calculated according to his capacity to hear speech, but if a standard for measuring loss of capacity for hearing speech be set up, it must be based on the way the ear hears speech, and not upon the way someone thinks it ought to hear speech. The data would then be invaluable not only for selecting candidates for military service but as well for compensating veterans for loss of hearing during their military service. Moreover, it will be most useful in diagnosis and therefore in selecting men for special services wherein perfect hearing is not essential. For example, a man suffering from obstructive deafness would function well in the heavy artillery and noisy factories. In such surroundings he would run less risk from acoustic trauma than would others. Why not, then, conserve hearing and man power by providing service for such men? Special provisions can be made for branches of the service where special hearing ability is essential, such as aviation, anti-aircraft, submarine detection, and so forth.

I suggest the following standards for the military services (see Table I).

TABLE I.

SUGGESTED HEARING STANDARDS FOR THE MILITARY SERVICES.

For Regular	Weighted difference between the two ears not more than
Army and Navy: less than a 5% loss in binaural capacity to hear speech	20 dcb.
(During Service): less than a 14% loss in binaural capacity to hear speech	30 dcb.
Aviation:	
Class I: less than a 5% loss in binaural capacity to hear speech	10 dcb.
Classes II and III: at least 5% but less than a 21% loss in binaural capacity to hear speech	No loss over 20 dcb. in important frequency bands

In pilots slight conduction deafness protects the ear from acoustic trauma and cuts down environmental noise. Tests should be made to determine the exact limitation to be set up. I have not had an opportunity to determine standards for such a specialized group.

For National Guard and Reserves:	Weighted difference between the two ears not more than
less than a 5% loss in binaural capacity to hear speech	30 dcb.
Selective Service:	
Class 1-A: (a) less than a 5% loss in binaural capacity to hear speech	20 dcb.
(b) at least 5% but less than 15% loss in binaural capacity to hear speech	30 dcb.
Class 1-B: (a) 15% or more but less than a 25% loss in binaural capacity to hear speech	(one ear may be totally deaf)
Class 4: (a) Loss of hearing capacity of 25% or more	

For old soldiers (officers and enlisted personnel) these standards may be somewhat lowered (the present custom is to ease up in this way).

In order to set up just criteria, it is essential to have a precise method of measurement which can be reproduced by other observers. The audiometer properly used provides this; the voice, whisper, watch tick, etc., do not.

The objection that the pure tone audiogram is not a true criteria for speech is not basically valid. The difficulty is not with the audiogram, which is admittedly a precise measurement of threshold, but with its usual misinterpretation through the unwarranted practice of averaging the speech tones, or multiplying by 0.8 and calling the result a percentage. Herein lies the fallacy. The practice not only assumes that all frequencies within the speech range have the same importance for the interpretation of speech but that no allowance is necessary for the changing increment of loss with increasing deafness. Such assumptions are definitely not warranted by the physiological facts underlying the hear-

ing of speech. All speech interpretation tests show the extreme importance of the middle and higher sounds (like S, th and ch), which like most of the consonants are weak and high pitched, whereas in comparison, the vowels are louder and lower pitched.

Some otologists are unable to understand how one may obtain better criteria for estimating percentage of loss of capacity for hearing speech with a machine such as a pure tone audiometer than with actual speech. Although at first thought one might think it would be easier to obtain a percentage by testing with speech directly, careful experiments show speech tests are very unreliable; they may vary as much as 200 per cent in distance even with trained examiners.* On the other hand, the audiometer is known to be exact, and observations made with it can be repeated with great accuracy. It is therefore better to test with the audiometer and use this data to interpolate for the capacity to hear speech than it is to use speech itself.

It is pertinent here to point out that decibel losses, or averages of decibel losses, being based on a geometric (logarithmic) scale are not as such translatable into a percentage loss which is arithmetic. A change from 30 dcb. to 40 dcb. is not the same change as is 40 dcb. to 50 dcb. The difference is not 10 per cent in either case, although in the second instance it is 10 times what it is in the first instance. However, this difficulty may be overcome if the audiogram is interpolated into percentage of loss of capacity for hearing speech.

* * *

A heretofore insurmountable difficulty has been the fact that nerve deafness is always accompanied by a "recruitment of loudness" (see discussion) for sounds over threshold, while conduction deafness is not;[1] *i.e.*, loud sounds appear of greater intensity to a person with nerve deafness than to a patient with conduction deafness. This fact accounts for the ability of one person to hear speech at a different distance than another, in spite of the fact that the air conduction threshold audiograms in both instances might be the same, and therefore the weighted or unweighted averages for the

*C.C. Bunch, personal communication.

speech tones would be absolutely the same. No further proof should be necessary to show the necessity of including the recruitment data in our calculations.

The following items are essential in the construction of a percentage standard for hearing speech, both in civil and in military life.

1. The use of the audiometer (in a soundproofed room) because it is the only dependable instrument at present available.

2. Weighting of the various frequencies according to their importance for hearing speech (see Table II) because if this is not done, injustices will occur, especially in instances of high tone losses.

TABLE II.

A METHOD FOR ESTIMATING THE PERCENTAGE LOSS OF CAPACITY TO HEAR SPEECH.

Weightings for the Frequencies Most Useful for Hearing Speech.

According to	250	500	1000	2000	3000	4000	8000*
Fletcher	3	7	40	40	0	10	0
Steinberg	2	15	20	34	0	26	3
Fowler "B"	2	15	30	35	0	15	3
"D"	0	15	25	30	25	5	0
"G"	3	15	20	30	20	10	2

*To save time and confusion the frequencies are given in the simplest numerals nearest to the usual octave designations.

3. Weighting for nerve deafness by the use of bone conduction measurements, as well as air conduction measurements. This will make adjustments for the "recruitment phenomenon" and prevent unfair discrimination between those suffering from varying degrees of obstructive as contrasted with nerve deafness.

4. The construction of a table which weights the percentage of loss according to the degrees of deafness. A table which will permit even a layman to figure the percentage of loss of capacity for monaural and binaural deafness.

A table which includes the important factors and excludes the nonessentials, a table which does all the hard work, so that all he has to do is first multiply, add and subtract to obtain the weighted loss for each ear and then apply these figures to the table to obtain the answer.

The answer thus obtained is more nearly correct than any he can obtain from any other method so far suggested. This statement is based upon a study of a large number of cases.* It is not necessary for him to understand the underlying facts in physics and physiology upon which the method is based.

In spite of the common opinion to the contrary, clinical observations are dependable, and quite as logical as standards evolved from precise physical data. All physical standards are based on assumptions.

TABLE III.

THE RECRUITMENT FACTOR (R. F.) IN NEURAL DEAFNESS.
(A Simplified Compilation of Clinical Observations by the Author.)

	Allowance
Up to 15 weighted dcb. loss for B. C.	0
Over 15 to 20 weighted dcb. loss for B. C.	—4
Over 20 to 25 weighted dcb. loss for B. C.	—3
Over 25 to 30 weighted dcb. loss for B. C.	—2
Over 30 to 32 weighted dcb. loss for B. C.	—1
Over 32 to 33 weighted dcb. loss for B. C.	0
Over 33 to 35 weighted dcb. loss for B. C.	+1
Over 35 to 40 weighted dcb. loss for B. C.	+2
Over 40 to 45 weighted dcb. loss for B. C.	+4
Over 45 to 50 weighted dcb. loss for B. C.	+8
Over 50 weighted dcb. loss for B. C.	+10

In moderate nerve deafness after an initial sharp recruitment of about 3 to 4 dcb. for sounds between 15 to 20 dcb. over threshold, there is a gradual lessening in the increment of recruitment of articulation understanding. Steinberg and Gardner show this clearly in their graphs.[2] When the weighted loss by bone conduction reaches 32 to 33 dcb. there is a reversal in the effect of the articulation recruitment phenomenon. Instead of being a minus (—) factor (an aid), it becomes a plus (+) factor (a detriment).

The allowances made for these + and — factors are clearly set forth in the above table, "The Recruitment Factor in Neural Deafness." Until the profession and the public are more familiar with the measurements of recruitment, the bone conduction weighted decibel losses may be used to determine them. The recruitment factor is added to or subtracted from the A.C. weighted dcb. losses in the right and left ears. The sum or the remainder, as the case may be, constitutes the weighted and adjusted decibel loss in each ear.

*Every case I examine is handled in this way.

The weighted decibel loss, either alone or adjusted, does not always give an accurate picture of hearing capacity. To accomplish this monaural and binaural allowances must be made for the different degrees of deafness. Tables IV and V do this.

DISCUSSION OF THE FACTORS UNDERLYING THE METHOD.

Hearing acuity may be measured in various ways. The most common are by tuning forks, the actual or recorded spoken word, or by the audiometer. Before attempting to set up a disability table for hearing loss the exact purpose and limitations of the method of choice should be stated, because it is impossible to calculate an over-all disability which would be fair to all classes and grades of the population. It is impossible to estimate with accuracy all the factors involved, such as the background of inheritance, social and educational training, occupation, language training, physical and mental stamina, attentiveness and urge to succeed. Variations in these backgrounds account for the fact that two people may have identical amounts and types of hearing loss and yet one may be very much more handicapped than the other.

The method I am here proposing is one for the estimation of the percentage of capacity for hearing speech (P.C.H.S.). The frequencies most useful in speech extend approximately from 250 to 8,000, but those near the extremes, 250 and 8,000, are relatively unimportant as compared with the mid-frequencies, so that it is usually rational and time saving to use only 500, 1,000, 2,000, 3,000 and 4,000. Several weightings for these speech frequencies are shown in the table. The weighting of choice depends upon the pitch and articulation characteristics of the voice in question, but a fair average is shown by the easily applied "D" weighting.

Weighting must also be employed for the "recruitment" phenomenon. This is shown in Tables II and III. Although recruitment of loudness is great for high degrees of nerve deafness, the nerve deafness contributes to the elevation of the threshold and therefore large volumes of sound are necessary to reach high thresholds, and this, with loss of clearness in articulation, limits the advantages gained from the "recruitment." On the other hand, recruitment for both loudness and articulation in slight nerve deafness is very rapid and efficient in clearness, quality, etc. In nerve deafness the recruitment factor is often equally if not more important than the frequency distribution.

In setting up standards, the first requisite is to define just what one proposes to measure and the meaning of the terms used.

I use the term "weighted decibel loss" to indicate the loss for the frequencies most useful for hearing speech, weighted according to their importance in hearing speech.

The audiogram is universally thought of as a threshold plotting of the different frequencies but such a conception does not necessarily measure what a patient hears, unless the frequencies are weighted for speech, and for the "recruitment factor" (R. F.) *Unless this is done it shows only what he cannot hear.*

To determine how much he can hear, it is necessary to measure the hearing for sounds well above threshold. This is the only sure way to measure the capacity for hearing speech. It is one of the basic factors which should always be included in audiograms and standards set up for estimating speech hearing loss.

By "recruitment" is meant the restoration of loudness which always occurs in nerve deafness areas as the intensity of sound is increased. Unfortunately, although the loudness may approach normal, the interpretability of speech does not increase proportionately so that it is necessary to apply Table III.

In the government services, as well as in civil life, it is not only the degree of deafness which causes varying disabilities but the effect on a man's ability to carry on in his particular occupation. Beasley's statistics show that the loss of income from deafness is progressively greater in the jobs of the higher mental attainment, clerk, managerial, executive positions, than in the lower grades, such as laborers.

Other things being equal, a child in the process of learning language is disabled more than an adult who has fully acquired language prior to his deafness.

A piano tuner will need to hear all the frequencies that he has to attune. To understand speech, the higher tones of the piano need not be heard. An expert draftsman may suffer little or no loss of income because of his deafness.

The ability to interpret speech varies to such an extent that all methods using actual speech are not really tests for loss of capacity to hear speech but rather tests for the ability to understand what is said (to interpret speech).

Irrespective of ability, training, etc., every hearing ear possesses a certain capacity for sensing the impulses necessary for the understanding of speech, and I here show how this can be measured audiometrically and translated into the percentage loss of capacity suffered from varying degrees of loss.

The speech hearing capacity may be determined by air conduction, by bone conduction, or best by both of these combined. Also by estimating the per cent of articulation, achieved at various distances or intensities, using syllables, words, phrases or sentences. All word tests introduce factors which confuse the results more than simple sounds like pure tones.

Bone conduction is important because it usually mirrors the "recruitment" to be expected in a given case. This phenomenon is important in nerve deafness. It may be an asset or a liability, depending upon the degree of neural involvement and upon whether or not the nerve deafened ear is masked by environmental noise.

Air conduction is usually the most important and satisfactory mode of hearing speech. One reason for preference is its steriophonic effect with binaural hearing. (Bone conduction is not so useful binaurally.) A loss in one ear cuts down the steriophonic effect and the general hearing capacity in a quiet place, depending upon the degree of loss in the opposite better ear and the difference between the two ears. In other words, a further monaural 10 dcb. loss over a prior binaural 25 dcb. loss causes a larger increase in percentage of loss than when the same ear goes down from such a 10 dcb. difference in loss to a further 10 dcb. loss. (A total of 20 dcb. difference from the opposite ear.)

Monaural losses cause more incapacity in noisy than in quiet places, because the opposite or better ear is made less efficient when masked by the noise. There is, then, temporarily in fact a binaural deafness in one ear caused by the disease, and in the other ear by the noise.

In a quiet room where the noise is only 15 to 20 dcb. in loudness, an ear that is down that amount by neural deafness is handicapped little, if at all, because conversation which is expected to be heard is usually well above 20 dcb. over threshold. The 20 dcb. noise if in the speech frequencies is therefore entirely masked, and is not forced upon the consciousness. It is not noticed as a detriment to the hearing of speech because the recruitment of loudness and of articulation increase rapidly for nerve deafness losses under 20 dcb. and so bring the speech up to approximately normal intensities.

TABLE IV.

TABLE FOR ESTIMATING PERCENTAGE OF LOSS OF CAPACITY FOR HEARING SPEECH.

(Monaural and Binaural Deafness.)

Worse ear — difference in wgtd. decb. loss	Weighted Decibel Loss (Better Ear)																							
	0	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100	105	110	
0	0	0	0	2	5	9	14	20	27	35	44	53	62	70	77	83	88	92	95	97	98	99	100%	Deafness for speech
10	0	0	2	....	8	....	18	....	32	....	49	....	66	....	80	....	90	....	96	....	99%			
20	0	1	3	....	10	....	21	....	36	....	53	....	69	....	82	....	91	....	97%	98				
30	1	....	4	....	11	....	23	....	39	....	56	....	71	....	83	....	92%	95						
40	2	....	5	....	12	....	24	....	41	....	58	....	73	....	84%	88								
50	3	....	6	....	13	....	25	....	42	....	59	....	74%	79										
60	4	....	7	....	14	....	26	....	43	....	60%	67												
70	5	....	8	....	15	....	27	....	44%	52														
80	6	....	9	....	16	....	28%	36																
90	7	....	10	....	17%	22																		
100	8	....	11%	13																				
110	9%	10																						

TABLE V.

TABLE FOR ESTIMATING PERCENTAGE OF LOSS OF CAPACITY FOR HEARING SPEECH.

(Monaural and Binaural Deafness.)

Equal losses both ears	Weighted Decibel Loss (Better Ear)																									
	0	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95	100	105	110			
0	0%																									
10	0	0	0%																							
20	0	....	2	2	5%																					
30	1	....	3	....	8	9	14%																			
40	2	....	4	....	10	....	18	20	27%																	
50	3	....	5	....	11	....	21	....	32	35	44%															
60	4	....	6	....	12	....	23	....	36	....	49	53	62%													
70	5	....	7	....	13	....	24	....	39	....	53	....	66	70	77%											
80	6	....	8	....	14	....	25	....	41	....	56	....	69	....	80	83	88%									
90	7	....	9	....	15	....	26	....	42	....	58	....	71	....	82	....	90	92	95%							
100	8	....	10	....	16	....	27	....	43	....	59	....	73	....	83	....	91	....	96	97	98%					
110	9	10	11	13	17	22	28	36	44	52	60	67	74	79	84	88	92	95	97	98	99	99	100%	Deafness for speech		

Weighted dcb. loss — worse ear.

In a given instance, to obtain the percentage loss of capacity, draw a vertical (ordinate) line from the figure representing the weighted decibel losses in the better ear, then draw a horizontal (abscissa) line from the figure representing the weighted decibel loss in the worse ear. The percentage of loss of capacity for hearing speech from monaural or binaural deafness will be found at the point where these vertical and horizontal lines intersect.

EXPLANATION OF TABLES IV AND V.

The table is constructed primarily for estimating the disability resulting from defective hearing for speech. The figures at the top of Tables IV and V represent the weighted decibel losses in a better ear. The figures on the left margin of Table IV represent difference in weighted decibel loss in the worse ear. In Table V, the left margin figures represent the weighted decibel losses in a worse ear. To facilitate computation, the figures representing losses in the better ear are shown in 5 dcb. steps, and in the worse ear in 10 dcb. steps, but the intervening steps should, of course, be utilized when the loss for an ear lies between the shown steps. Table V is simply another arrangement of Table IV.

The percentage loss of capacity for speech extends from perfect hearing (0 loss) to total deafness for speech (100 per cent loss) (not necessarily total deafness for all sounds). These are, of course, the extremes obtainable. A 50 dcb. weighted loss in both ears is estimated to produce a 44 per cent loss in capacity for hearing speech. Between these extremes and this mean, the table is constructed according to the varying increments and decrements of loss of capacity with increasing weighted decibel losses.

The percentages of loss of capacity are given in the body of the table. In this case, also, the intervening steps should be used. The percentage figures are based on the average loss of capacity clinically observed in people suffering from various degrees and types of deafness. The clinical test is always the final answer as to reliability.

Under ordinary circumstances, there should be no difference in estimating the loss in hearing capacity whether the right or the left ear is the one more affected.

The relative differences between the figures representing the varying percentage losses in bilaterally equal and in bilaterally different ears are necessary because the worse ear becomes progressively less important as it loses capacity.

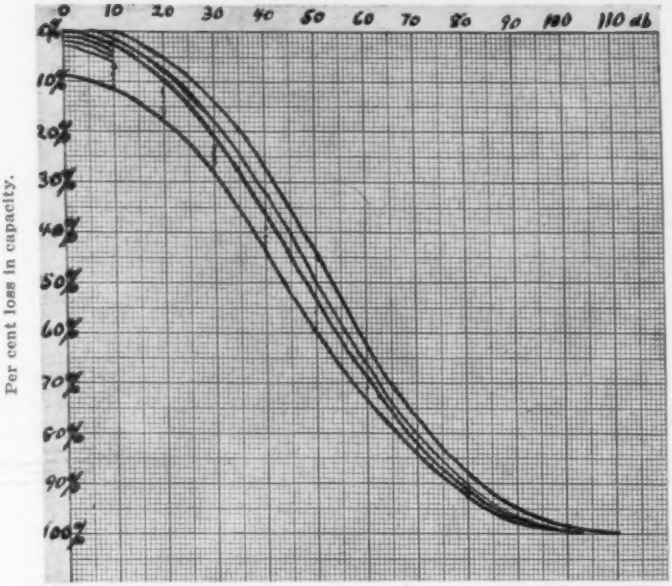
If one ear is totally deaf, the remaining hearing ear (lone ear) inflicts a greater and up to 50 dcb., a more rapid general loss of capacity from increasing deafness than two similarly hearing ears. For more than a 50 dcb. loss the lone ear loses capacity at a lesser rate than two equally hearing ears but, of course, at a lower level. (The table and the graph show this.)

It must be kept in mind that a good ear is in a position to hear sounds from the totally deaf side over two-thirds of the time because it is aided by anticipation of the sound source through the other senses, or by aids, so that the head is turned to enable the hearing ear to hear the sounds. However, there is no spare ear if the hearing in the one and only hearing ear becomes impaired, and warning sounds from the deaf side are often not heard.

My tests appear to prove that even a totally deaf ear increases the handicap only about 10 per cent unless the hearing ear is below average normal.

Clinical observations show that up to a 20 dcb. weighted binaural loss, there is little loss in capacity. However, 2 per cent is assigned to a 15 dcb. binaural loss, and 5 per cent to a 20 dcb. binaural loss. From there on up to a 50 dcb. weighted loss the handicap mounts with increasing increment of loss. (One per cent is added to each lower 5 dcb. weighted loss.) Above 60 the percent-

GRAPHS OF TABLES IV AND V.



Distances along ordinates represent per cent loss in binaural hearing capacity from 10 dcb. differences in worse ear.

Upper curved line represents per cent loss in equal binaural deafness.

The next line represents the per cent loss when there is a 10 dcb. difference between the better and the worse ears.

The third line from the top represents the per cent loss when there is a 20 dcb. difference between the two ears.

The dots below this third line represent similar lines at intervals of 10 dcb. (omitted for clarity).

The lower curved line represents per cent loss in a lone hearing ear (other ear totally deaf).

age of capacity loss increases, but with a decreasing increment as the deafness increases. (One per cent is subtracted from each lower 5 dcb. weighted loss.) Incidentally, this has to be done in order that the percentage of loss can taper off toward the total deafness end of the scale. In this connection it is pertinent to observe that a person with even a remnant of capacity, say 95 per cent, is certainly a little better off than a person with no capacity to hear speech. For ease of computation, all the percentage figures are shown as whole numbers.

A 110 dcb. weighted loss is practically useless for hearing speech without artificial aids and therefore no allowance is made for *(continued on page 952)*

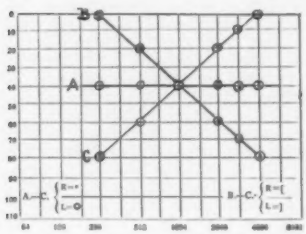


Fig. 1. If the ears of three people measure at thresholds (binaurally equal) as shown in the curves A, B and C, all will average 40 dcb. loss unless the frequencies are weighted, and yet these three people will not suffer equal losses in capacity to hear. Assuming bone conduction to be normal, and weighting the speech frequencies (approximately 250, 500, 1,000, 2,000, 3,000, 4,000 and 8,000) according to the Fowler "G" weighting figures, the result for both ears in (A) will be a 40 dcb. weighted loss; for both ears in (B) a 53 dcb. weighted loss; and for (C) a 27.4 dcb. weighted loss.

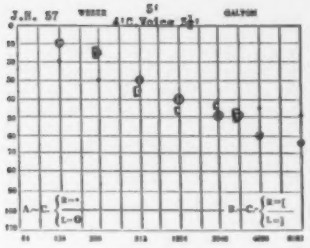
Using Table V, the binaural loss in capacity measures for (A) 27 per cent, for (B) 50 per cent and for (C) 11 per cent. These differences are brought about because weighting is properly distributed more to the higher than to the lower speech frequencies.

When (A) and (B) are drawn to represent a theoretically typical nerve deafness, with bone conduction coinciding with air conduction, the hearing in both instances will be worse than appears from the threshold air conduction graphs alone. The nerve deafness and the recruitment (rect.) factor are the cause of this. In (A) add the recruitment factor (plus 2) to the weighted decibel loss; in (B) add 10, as indicated in the computation chart. Case A will then be shown to suffer a 30 per cent loss, and (B) a 67 per cent loss in capacity. This is just about what people with similar losses actually do suffer.

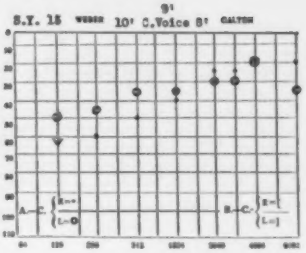
Theoretical Audiograms Showing Three Types of Hearing Losses.

Frequencies	Dcb. Loss A Wgtg.	Dcb. Loss B Wgtg.	Dcb. Loss C Wgtg.
250	40 x 3 — 120	0 x 3 — 0	80 x 3 — 240
500	40 x 15 — 600	20 x 15 — 300	60 x 15 — 900
1000	40 x 20 — 800	40 x 20 — 800	40 x 20 — 800
2000	40 x 30 — 1200	60 x 30 — 1800	20 x 30 — 600
3000	40 x 20 — 800	70 x 20 — 1400	10 x 20 — 200
4000	40 x 10 — 400	80 x 10 — 800	0 x 10 — 0
8000	40 x 2 — 80	100 x 2 — 200	0 x 2 — 0
	40.00	53.00	27.40
Using Table V % loss of capacity equals		27%	50% 11%
If B.C. in audiograms A and B coincide with A.C. add rect. fact. to wgtg. dcb. loss to obtain (see note)	2.00 / 42.00	and 10.00 / 63.00	the adjusted dcb. loss
The percentage loss in binaural capacity will then be		30%	67%

NOTE: If instead of weighting the bone conduction losses the same as the air conduction losses, they are weighted solely to obtain the recruitment factor at each frequency, and this factor is added to or subtracted from the A.C. dcb. loss at each frequency, the resultant calculations will correspond even more closely with actual clinical observations. The same "recruitment factor" figures may be used as indicated in Table III assigning —1 to losses of 35 dcb. instead of only to over 30 to 32 dcb. losses as in table. In the above audiograms A and B, if A.C. is identical with B.C. it would obviously make no difference which method of using the recruitment factor was employed in A, but a slight difference in B. From a strictly scientific standpoint "recruitment" should be measured, or estimated, for at least each of the five frequency areas most useful for speech, 500, 1000, 2000, 3000 and 4000.



2A



2B

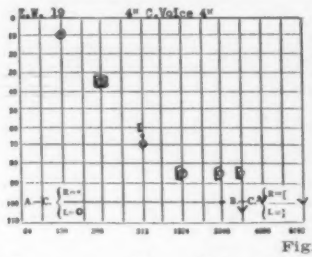
Fig. 2.

A.C. Right	A.C. Left	B.C.	
35 x 15 = 525	30 = 450	30 = 450	512
40 x 25 = 1000	40 = 1000	40 = 1000	1024
50 x 30 = 1500	50 = 1500	45 = 1350	2048
50 x 25 = 1250	50 = 1250	50 = 1250	3012
45 x 5 = 225	60 = 300	45 = 225	4096
45.00	45.00	42.75	
+4.00	+4.00	Rect. factor	
49.00	49.00	Adj. dcb. loss	

Table V "D" weighting 42% loss

A.C. Right	A.C. Left	B.C.	
50x15 = 750	35 = 525	Normal	
40x25 = 1000	35 = 875		
25x30 = 750	30 = 900		
25x25 = 625	30 = 750		
20x 5 = 100	20 = 100		
32.25	31.50		
0.	0.	Wgtg. dcb. loss	
		Rect. factor	
32.25	31.50	Adj. dcb. loss	

Table V "D" weighting 16% loss



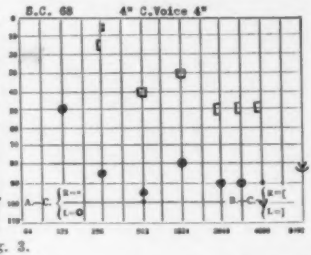


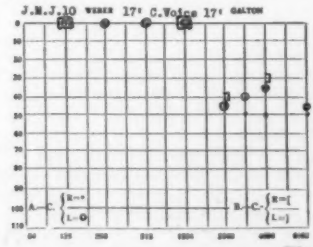
Fig. 3.

A.C.	Rt.	Left	B.C.
Wgtd. dcb. loss:	95.00	84.00	67.00
Rect. factor	+10.00	+10.00	
Adj. dcb. loss:	105.00	94.00	

Table V "D" wgting. 97% loss.

A.C.	Rt.	Left	B.C.
Wgtd. dcb. loss:	89.00	89.00	44.00
Rect. factor	+4.00	+4.00	
Adj. dcb. loss:	93.00	93.00	

Table V "D" wgting. 96% loss.



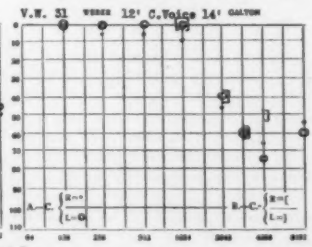


Fig. 3.

A.C.	Rt.	Left	B.C.
Wgtd. dcb. loss:	27.00	21.00	23.50
Rect. factor	—3.00	—3.00	
Adj. dcb. loss:	24.00	18.00	

Table V "D" wgting. 5% loss.

A.C.	Rt.	Left	B.C.
Wgtd. dcb. loss:	33.85	30.75	29.70
Rect. factor	—2.00	—2.00	
Adj. dcb. Loss:	31.85	28.75	

Table V "D" wgting. 13% loss.

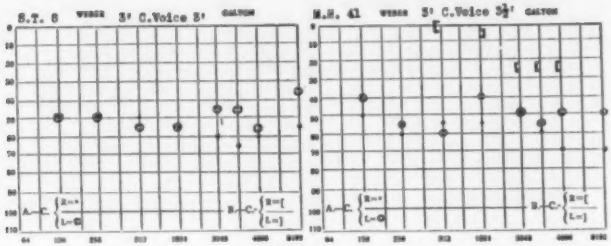


Fig. 4.

A.C.	Rt.	Left	B.C.	A.C.	Rt.	Left	B.C.
Wgtd. dcb. loss:	59.00	49.50	normal	Wgtd. dcb. loss:	55.50	50.25	17.75
Rect. factor	0.	0.		Rect. factor	—2.00	—2.00	
Adj. dcb. loss:	59.00	49.50		Adj. dcb. loss:	53.50	48.25	

Table V "D" wgting. 48% loss. Table V "D" wgting. 42% loss.

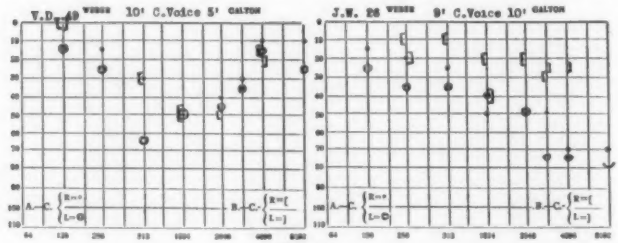


Fig. 4.

A.C.	Rt.	Left	B.C.	A.C.	Rt.	Left	B.C.
Wgtd. dcb. loss:	37.00	45.25	37.00	Wgtd. dcb. loss:	47.25	52.75	19.40
Rect. factor	+2.00	+2.00		Rect. factor	—4.00	—4.00	
Adj. dcb. loss:	39.00	47.25		Adj. dcb. loss:	43.25	48.75	

Table V "D" wgting. 28% loss. Table V "D" wgting. 34% loss.

losses over 110 dcb. A totally deaf ear is of no use to an opposite hearing ear (for hearing or for quality). A partially deaf ear is of some use to an opposite hearing ear because it often improves the quality and clearness of sounds and their localization in space (steriophonic effect).

Figures in the table are not absolute. They may with experience be changed but it will not be possible to vary them more than a few per cent up or down because the figures must be contained between zero and 100 per cent loss, and this is impossible to accomplish under the conditions if the figures are varied much.* In other words, dozens of trial tables have been constructed and I have been forced by the limitations of the hearing range (0 to 100) (and clinical

*Subsequent to presentation, the % figures were shifted 5 dcb. to the left, in order to avoid using the recruitment factor for deafness over 45 dcb.

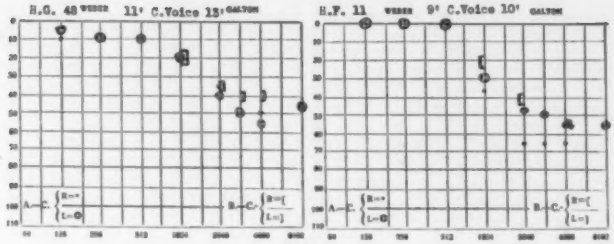


Fig. 5.

A.C.	Rt.	Left	B.C.
Wgtd. dcb. loss:	32.00	33.75	24.50
Rect. factor	—3.00	—3.00	
Adj. dcb. loss:	29.00	30.75	

Table V "D" wgting. 14% loss.

A.C.	Rt.	Left	B.C.
Wgtd. dcb. loss:	48.25	36.25	32.25
Rect. factor	+1.00	+1.00	
Adj. dcb. loss:	49.25	37.25	

Table V "D" wgting. 26% loss.

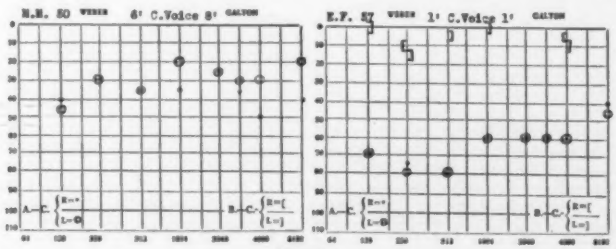


Fig. 5.

A.C.	Rt.	Left	B.C.
Wgtd. dcb. loss:	36.50	29.25	normal
Rect. factor	0.00	0.00	
Adj. dcb. loss:	36.50	29.25	

Table V "D" wgting. 15% loss.

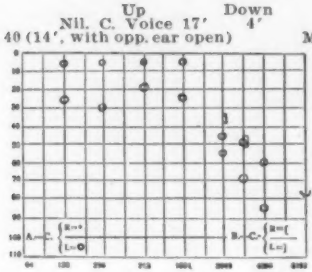
A.C.	Rt.	Left	B.C.
Wgtd. dcb. loss:	63.00	63.00	normal
Rect. factor	0.00	0.00	
Adj. dcb. loss:	63.00	63.00	

Table V "D" wgting. 67% loss.

experience) to adopt the figures shown in the tables for the bilateral equal loss of hearing. Whether or not the figures showing the percentage of loss of capacity from unequal bilateral hearing losses are final, any legitimate change in them would not make a great difference.

The ability or inability to use hearing aids is of real importance in the same way that eyeglasses or a crutch are important to those who cannot function well without them.

The ability to use the aid (as a crutch) certainly affects one's ability to function in the world, and earn a living, but this should not enter into the basic estimation of hearing capacity. It should enter into any just calculation of disability from the deafness. The standard I have set up enables one to accomplish this.



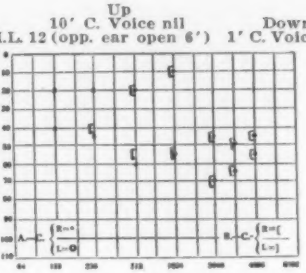
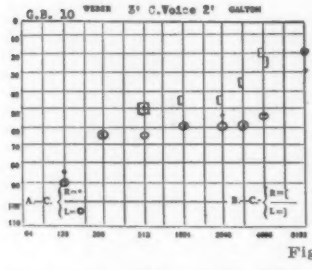


Fig. 6.

Up 24		Down 59		% loss.	Up 36		Down 87	
A.C.	Up	B.C.	Down	B.C.	A.C.	Up	B.C.	Down	B.C.
Wgtd. dcb. loss:	31.00	11.0	47.50	35.00	Wgtd. dcb. loss:	33.75	33.70	63.50	62.00
Rect. factor	—4.00		+1.00		Rect. factor	+1.00		+10.00	
Adj. dcb. loss:	27.00		48.50		Adj. dcb. loss:	34.75		73.50	

Table V "D" weighting.



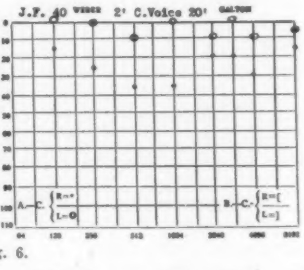


Fig. 6.

A.C.	Rt.	Left	B.C.	A.C.	Rt.	Left	B.C.
Wgtd. dcb. loss:	56.75	60.50	51.00	Wgtd. dcb. loss:	26.50	5.00	normal
Rect. factor	+10.00	+10.00		Rect. factor	0.00	0.00	
Adj. dcb. loss:	66.75	70.50		Adj. dcb. loss:	26.50	5.00	

Table V "D" wgting. 74% loss. Table V "D" wgting 1% loss.

It is often stated that because the visual acuity of the eye is expressed as a fraction that this represents a per cent of acuity, and that auditory capacity should be set up in the same manner. Of course, the fraction does nothing of the kind. The numerator and the denominator represent the number of feet the tested eye and the normal eye read the standard chart; 20/40 therefore does not mean a 50 per cent loss. The American Medical Association Committee worked out a table for estimating loss of eyesight which has been accepted by insurance companies and compensation boards in various States. In constructing this table the committee first estimated the decreased efficiency from

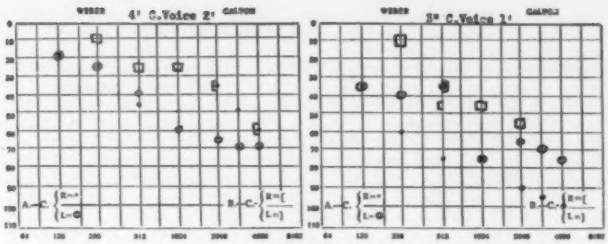


Fig. 7.

C.F. 15

Hearing up.				Hearing down.			
A.C.	Rt.	Left	B.C.	A.C.	Rt.	Left	B.C.
Wgtd. dcb. loss:	45.25	61.50	36.00	Wgtd. dcb. loss:	85.75	64.75	54.25
Rect. factor	+2.00	+2.00		Rect. factor	+10.00	+10.00	
Adj. dcb. loss:	47.25	63.50		Adj. dcb. loss:	95.75	74.75	

Table V "D" wgting. 47% loss. Table V "D" wgting. 88% loss.

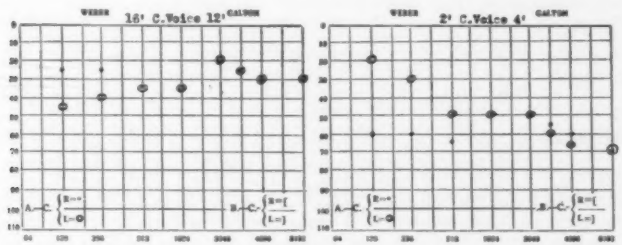


Fig. 7.

J.W.S. 33

Hearing up.				Hearing down.			
A.C.	Rt.	Left	B.C.	A.C.	Rt.	Left	B.C.
Wgtd. dcb. loss:	27.75	27.75	normal	Wgtd. dcb. loss:	54.00	53.50	normal
Rect. factor	0.00	0.00		Rect. factor	0.00	0.00	
Adj. dcb. loss:	27.75	27.75		Adj. dcb. loss:	54.00	53.50	

Table V "D" wgting. 13% loss. Table V "D" wgting 56% loss.

the loss of sight of one eye and then of two eyes, and set up an arbitrary standard. Total disability was used as the base line and corresponded to a complete loss of vision in both eyes. I have constructed my table to include comparable factors affecting the ear, and in addition several which if not included would enable any bright lawyer to tear it to pieces. In the case of the standards set up for the eye, some important factors have been omitted, and may unexpectedly throw a monkey wrench into the standards. The accompanying audiograms show varying degrees and types of deafness, and in each instance the per cent of loss in capacity to hear speech according to the standards I have set up (see Figs. 1 to 7). The distance the voice was heard is indicated above the upper, and below the lower audiograms, and in Table VI.

TABLE VI.

The Percentage of Loss of Hearing Capacity Compared with the Distance That Conversational Speech Was Heard in the Persons Whose Audiograms Are Shown in Figs. 2 to 6, Inclusive.

Conversational Voice Heard at				Table V "D" Weighting
Right	Left	Patient	Age	% Loss Capacity
2'	20'	J. F.	40	1%
17'	17'	J. M. J.	10	5%
12'	14'	V. W.	31	13%
11'	13'	H. G.	48	14%
6'	8'	M. M.	50	15%
10'	8'	S. Y.	15	16%
9'	10'	H. F.	11	26%
10'	5'	V. D.	49	28%
9'	10'	J. W.	26	34%
3'	3½'	M. H.	41	42%
4'	3½'	J. H.	57	42%
3'	3'	S. T.	8	48%
2'	4'	J. W. S.	33	53% (13% when hearing is up.)
4' (Lone ear)		H. K.	40	59% (24% when up.)
1'	1'	E. F.	57	67%
3'	2'	G. B.	10	74%
1' (Lone ear)		M. L.	12	87% (36% when up.)
3"	1'	C. F.	15	88% (47% when up.)
4"	4" (few words)	S. C.	68	96%
4"	4" (some words)	E. W.	19	97%

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140 East 54th Street.

MILITARY SYMPOSIUM.

MANAGEMENT OF INJURIES TO MIDDLE AND INTERNAL EAR INCLUDING FRACTURES OF THE TEMPORAL BONE.*

DR. W. E. GROVE, Milwaukee.

The membrana tympani may be ruptured in three ways:

1. Direct penetration by a foreign body.

2. Extension of a temporal bone fracture to the membrane.

3. Condensation or rarefaction of the air in the external auditory canal.

1. Direct penetration of the tympanic membrane may occur by the use of such things as hairpins, toothpicks, matches, straws, etc., to relieve itching; by careless instrumentation in the removal of a foreign body from the external canal; by flying foreign bodies such as chips of wood, splinters of glass, small shell fragments., etc.; by the penetration by a twig while the patient passes through a thicket, by hot molten metal entering the external canal, and, in some instances, by reason of a projectile piercing the membrane.

2. A basal skull fracture involving the temporal bone longitudinally may project itself through the middle ear, injure its contents, rupture the drum and fracture the walls of the external auditory canal. This is associated with a discharge of blood or cerebrospinal fluid from the ear. In such instances, as well as when the drum and middle ear are directly penetrated by a projectile, the rupture of the drum and the injury to the middle ear assume a secondary importance, being overshadowed by the craniocerebral damage produced.

3. Ruptures of the membrana tympani are caused by a sudden condensation or rarefaction of air in the external canal due to a box on the ear, a fall on the ear, the explosion of a large gun or the bursting of a shell in close proximity, sudden compression or decompression in caisson workers and among airplane personnel by sudden changes of altitude. In sudden changes of air pressure in the external canal, rupture

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of the drum is more apt to occur if the Eustachian tube is closed.

The internal ear may be injured by basal skull fractures which involve the temporal bone in a longitudinal or in a transverse fashion, by injuries to the head which cause a vault fracture without damage to the base, by injuries to the head which cause no fracture at all, by the concussion resulting from the explosion of a large gun or the bursting of a shell in close proximity, by the sudden condensation of air in the external auditory canal and by the passage of a projectile directly through the internal ear. In those cases in which the internal ear itself is fractured by a blow, fall or projectile, the usual ear symptoms are complete cochleovestibular paralysis, with or without facial paralysis. In longitudinal fractures of the temporal bone the usual ear symptoms are the escape of blood or cerebrospinal fluid from the ear, bleeding into the basal coil of the cochlea, and damage to the vestibular mechanism, with or without facial paralysis. In the injuries to the head which do not cause bone fractures the symptoms concern the hearing and vestibular function.

TREATMENT.

Traumatic ruptures of the drum not associated with basal skull fractures are usually irregular in outline. Some bleeding may occur but it is not excessive. These ruptures usually heal by first intension unless infection occurs. They should be treated conservatively and expectantly with sterile dressings applied over the ear. Middle ear suppuration is encouraged by repeated examination and ill-advised attempts to cleanse the external canal. If middle ear suppuration supervenes it is to be treated according to our time-honored methods of handling such suppuration, whatever its cause. The perforations of the drum caused by burns are much more stubborn and in these cases middle ear suppuration almost always follows after the subsidence of which persistent perforations of the drum often result. Ruptures of the drum caused by a sudden elevation of air pressure in the external canal rarely suppurate except as the result of bad management.

If, after an injury to the head which causes a temporal bone fracture and there is a discharge of blood or cerebro-

spinal fluid from the ear, the policy of absolute *"laissez faire"* should prevail as pertaining to the ear. Sterile cotton may be placed in the meatus and the ear covered with sterile dressings. Nurses and internes should be warned not to remove the dressings or manipulate the ear. After a few days the otologist may inspect the ear, but in so doing he should maintain a meticulous asepsis. If the ear is still bleeding, the sterile cotton and sterile dressings are replaced. Under no circumstances should such an ear be cleansed in less than two to three weeks, and even then mechanically with sterile instruments. Even at this date irrigation of the ear should not be practiced because of the possibility that a perforation of the membrana tympani may still exist. The practice of certain European otologists, which is followed by many physicians in this country, of cleansing the external canal with alcohol or peroxide, either by swab or by irrigation, should be condemned. Many cases of ear suppuration have most certainly been occasioned by such treatment. If the bleeding is so profuse as to lead to the conclusion that the sigmoid sinus has been injured, an extremely rare condition, one may have to expose and block the sinus. Certainly, packing the external canal of an ear which is bleeding will not stop the hemorrhage coming from within the middle ear through the ruptured drum, and may induce an otitis.

Naturally, when a basal skull fracture has taken place, the treatment of the craniocerebral damage is of prime importance, and that of the ear itself assumes a secondary place. Damage to the brain by pressure may be due to the fracture itself or to some type of hemorrhage. Hemorrhages in and about the brain may be classified under five general types.

1. Extradural hemorrhage, usually due to a rupture of the middle meningeal artery.

2. Subdural hemorrhage, usually due to a tearing of thin-walled sinuses.

3. Subarachnoid hemorrhage in which the blood spreads out over the cortex and a hemorrhagic pachymeningitis takes place.

4. Subcortical hemorrhage, usually petechial in form and, when occurring in the brain stem, the causative factor of vestibular symptoms.

5. Intraventricular hemorrhage.

We recognize three stages in patients with severe head injuries: *1.* the phase of shock; *2.* the phase of increasing intracranial pressure; and *3.* the phase of medullary failure.

The phase of shock is manifested by the usual symptoms accompanying surgical shock; the stage of increased intracranial pressure is manifested by a rising blood pressure and a falling pulse; in the stage of medullary failure the blood pressure falls and the pulse rises.

In recent years the general treatment of severe craniocerebral injuries has taken a very conservative trend, except in isolated cases. The treatment of the shock should take precedence over everything else. The suture of wounds, detailed neurological examination, X-ray examination and any unnecessary movement of the patient should be delayed until adequate recovery from shock has taken place. Shock is treated by shock cabinets, warm dry blankets, heat to the extremities, and elevation of the foot of the bed. Appropriate medication such as atropine, pituitrin, ephedrine, ergot or strychnine may be used.

After the stage of shock, further treatment may be medical or surgical. The indications for operation are: *1.* compound fractures of the skull, or fractures with marked depression; *2.* cases of extradural hemorrhage, for which condition the development of unconsciousness after a lucid period is almost pathognomonic; and *3.* cases of increased intracranial pressure not improving under medical treatment and where signs of definite localization such as paralysis or convulsions appear.

The medical treatment may be outlined as follows:

1. Rest in bed, which should be absolute for three to six weeks.

2. Posture. The head and trunk should be elevated.

3. Drugs. Caffeine sodium benzoate has a distinct tendency to lower intracranial pressure. Restlessness can be controlled by bromides and the barbiturates. Morphine is contraindicated because it tends to increase intracranial pressure.

4. Dehydration can be obtained by the use of magnesium sulphate, hypertonic solutions of sucrose or glucose given

intravenously, and by the limitation of the fluid intake to 800 or 1,000 cc. per day.

5. Repeated lumbar punctures serve to lower the intracranial pressure and to remove blood from the subarachnoid spaces.

INTERPOLATION.

This has more to do with primary surgery, and is not primarily otological, but is included for the benefit of those in our specialty who may be called upon to take care of such injuries and not have the benefit of the general practitioner or surgeon.

THE EAR SURGERY OF TEMPORAL BONE FRACTURES.

In general, it may be said that most otologists adopt a very conservative attitude toward any surgery of middle ear or mastoid in fractures involving the temporal bone. Notable exceptions to this statement are Voss, Linck and a few others. In most cases in which blood or cerebrospinal fluid discharges from the ear after a longitudinal fracture of the temporal bone such discharge ceases in a few days and the drum closes in a few days or weeks, unless there have been ill-advised efforts to cleanse the external auditory canal or pack it.

The classical rule in the treatment of longitudinal fractures has been to avoid any surgery unless the fracture was complicated by ear and mastoid suppuration. More recently, the school of Voss and Linck has advocated active surgery in all types of temporal bone fractures, but the large majority of otologists are still inclined to conservatism in this regard.

If an otitis media develops in ear which has been draining blood alone, it should be treated as any other acute middle ear suppuration. It should be treated in the same manner as though no fracture existed. Chemotherapy may be used if desired, but the indiscriminate use of these chemotherapeutic drugs, which of themselves have a tendency to produce a hemolytic anemia, should be condemned in a patient who has suffered from shock and loss of blood.

If the longitudinal fracture produces a discharge of cerebrospinal fluid from the previously healthy ear, such a dis-

charge indicates a connection between the subarachnoid space and the exterior via the middle ear. These cases may also be treated expectantly and conservatively. The vast majority of them heal without infection. Should an otitis media supervene, a simple mastoid operation will produce adequate drainage. Intensive treatment with chemotherapeutic drugs should then be instituted and the case watched for any signs of meningitis.

Should the longitudinal fracture occur through a chronically suppurating ear, the indications are somewhat different, for in this case a real danger of meningeal involvement exists. Such a case should be operated upon as soon as the period of shock is over. A radical exposure should be made, the fracture line followed and exposed as far as possible, the wound left wide open and intensive chemotherapeutic therapy instituted.

The question of operative interference in labyrinthine fractures, and more particularly in labyrinthine fractures which are also associated with longitudinal fractures, is somewhat different than in the case of pure longitudinal fractures. This question is bound up with the fact that in about 50 per cent of fractures of the otic capsule the lateral labyrinth wall is fractured and the labyrinth put into direct communication with the middle ear. As microscopic examination teaches us that these capsular fractures heal by fibrous union and are never completely closed by a bony callus, the danger of late meningitis always exists. If a post-traumatic otitis develops in this sort of a case, even many years after the injury, the danger of meningitis is great. While a few such otitides recover under conservative treatment, the consensus of opinion among otologists is that such a late otitis should receive radical surgical care. With the concomitant administration of sulfanilamide or sulfapyridine, the prognosis of such a condition should be greatly enhanced.

Facial paralysis develops in 10 to 20 per cent of the cases of longitudinal fractures and in as high as 50 per cent of the cases of fractured otic capsule. These paralyses may be immediate in onset or delayed. When immediate, they are due to actual injury to the nerve, either in the Fallopian canal or at the internal auditory meatus. The prognosis for such immedate paralysis is dubious. In the delayed variety the prog-

nosis for recovery is much better. Surgery on such paralyzed nerves is not indicated, for when the paralysis is immediate, the injury usually affects the nerve at or internal to the knee, and this region is not amenable to surgical attack. Recovery is the rule in cases of delayed facial paralysis.

Unless the longitudinal fracture, in its passage through the roof of the tympanum, actually damages the ossicular chain, the damage to the cochlear function is of the perceptive type and is due to hemorrhage into the basal coil of the cochlea. Such hemorrhages are in the perilymph system and may partially or completely absorb. If they do, the defect of hearing is not marked. In other cases in which the hemorrhage is not absorbed, one may expect a gradual further deterioration of hearing caused by the laying down of connective tissue and bone in the perilymph spaces. Such hearing defects are not amenable to treatment.

The vertigo caused by direct invasion of the labyrinth usually subsides in a few weeks under bed rest, dehydration and sedation. It is usually more or less continuous in character. If it persists after this period, it is usually intermittent in character and is due, in my opinion, to vasomotor changes in the brain stem induced by petechial hemorrhages and areas of focal necrosis in this area. As this type of vertigo is increased by sudden movements of the head, exposure to heat, indulgence in alcohol and elevation above ground level, the patient should be cautioned to avoid these things. The best treatment for this symptom, as well as for the post-traumatic headache, is the dehydration and prolonged bed rest after the injury. After the patient has been released from the hospital, the symptom can be best controlled by sedation with bromides or the barbiturates. In a few instances I have had very good success in controlling the vertigo and the headache by the histamine treatment as outlined by Sheldon and Horton, which convinces me that, in some instances at least, these post-traumatic symptoms are vascular in origin. In some cases these symptoms gradually disappear, while in others they remain unabated for years. After a reasonable period of convalescence these patients should be encouraged to return to some form of light work, for otherwise they are prone to develop post-traumatic neuroses and psychoses.

324 East Wisconsin Avenue.

MILITARY SYMPOSIUM.

PREVENTION AND TREATMENT OF ACOUSTIC TRAUMA IN WAR AND CIVIL LIFE.*

DR. DOUGLAS MACFARLAN, Philadelphia.

I shall try to confine myself to the exact title of the paper — "Prevention and Treatment of Acoustic Trauma." Yet, I regret that the causes of this trauma will frequently have to be mentioned. Already, we have heard the paper of Dr. Grove on the "Management of Injuries to the Middle and Inner Ear"; and we are still to hear from our two military guests.

I would draw your attention to a fairly full literature on the subject, and have subtended a bibliography that may be of service. The subject has surely not been overlooked.

Prevention, of course, suggests the correction or abolition of the noises and concussions that cause trauma. In war, this is largely impossible; in civil life much can and will be done. Like the worry we have in obtaining so-called sound-proof rooms, our thoughts turn to reducing the noise at its source. The replacement of the riveter by the electric welder is an advance along the lines desired. Modernization has also developed many inventions to reduce friction and jar — bearings and moving parts, such as wheels, gears and springs, are now designed with the thought of cushioning and smoothing out motion, usually accomplishing a conservation of force, a lessening of wear and a reduction in noise. Thus, the usual noises of manufacturing and transportation may be reduced — an important thing for not only our hearing but for our nerves. (Consult the report of the New York Committee on Noise Abatement[1] for the importance and correction of commonplace noise.)

In war, nothing can be done about shell and bomb explosions, nothing can be done about the noise when one is inside a moving tank, and men will have to leave the engine room of the mosquito boats when the twin Packard motor is

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started. It is possible to do much, however, in the protection against "outgoing" noise. The rifleman can be warned not to get ahead of the man firing next to him, the artillery man can be trained to stuff up his ears and keep his mouth open. As to ear plugs — it was hard to get the infantryman in the last war to wear plugs; he naturally wanted to hear well when the excitement was on, and not without reason. Though it was much better for a soldier to be wearing plugs when shells were bursting nearby, it was equally important, he felt, that he hear the "spud" of machine gun bullets hitting in the mud close to him, and he wanted also to hear the commands and shouts of those about him.

To protect civilians against trauma in air raids, plugs are of great value. Fifty million gutta-percha ear plugs were ordered by the British Ministry of Home Security for distribution to the public. An old lady received a pair — they were marked "R" and "L" and instructions for inserting them were sent with the parcel. The old lady not only wrote a letter of thanks to the donor but also wrote a note of appreciation to the makers, in which she said, "My hearing has improved beyond belief since I have been wearing your excellent plugs."

Larson[2] reminds us that closing of the ears is considered of little value (or importance) by the laborers in noisy occupations. This can be explained rather on the grounds of laziness, indifference, inconvenience and ignorance. Persons with labyrinthine symptoms or affections, tinnitus, dizziness and smaller degrees of nerve deafness, as well as those with chronic middle ear suppuration, should not be exposed to loud noises, either in civil or military life.

Yet, where no serviceable hearing remains, we may well choose the noisiest occupation for certain of these cases, as Dr. Fowler suggests in his paper today.

An interesting comment can be made on the possibility of acoustic trauma when the amount or rate of fire of some of our modern weapons is considered. With repeated stimuli there is repetition of latency periods, during which acoustic oscillations of the conducting mechanism are undampened and unimpeded and the end-organ is violently stimulated. Animals exposed to frequently repeated loud sounds show evi-

dence of damage to the perspective mechanism sooner than do similar animals exposed to a continuous, though equally loud, sound stimulus — the ear unprotected by the stapedius muscle fatigues more easily.

The latent period of the stapedius is 10.5 milliseconds.[3]

The Gerand .30 calibre rifle fires a clip of eight shots in a few seconds when used as an automatic. With average skill, I believe, six to eight clips can be fired in a minute.

The .37 millimetre fires 30 per minute.

The .30 calibre light air-cooled machine gun fires 425 rounds per minute.

The .30 calibre water-cooled machine gun fires 650 rounds per minute.

The .50 calibre gun fires 375 to 475 shots per minute.

Though the bursts of this fire are not as concussive as the "heavies," the aggregate summation of acoustic shock is serious.

The same is true of aeroplane engine noise — on which subject there is an excellent review by Dr. Crowe[4] of Dickson, Ewing and Littler's paper on the study of the present Royal Air Force. After a few hundred hours' flying without a helmet in an enclosed cockpit, the results of hearing tests show high tone deafness. The individuals engaged in testing and tuning also show this loss, first appearing as an abrupt dip at 4,000 cycles. In the beginning of the flying career, some of this hearing loss may be recovered if the aviator gives up the service, but with continued service the deafness fixes itself; in fact, gets progressively worse, until it creeps down upon the speech frequency zone. Aural fatigue is thought to be greatest at or about the frequency of the fatiguing tone, but studies of aeroplane noise indicate that all of its components of large amplitude are low pitched and occur below the frequency of 700. Thus, it appears that low pitched noises cause high tone deafness. As Guild and others have shown in these cases, there is degeneration in the basal turn of the cochlea.

Here is an interesting slide which Dr. Lurie has given me. It shows the organ of Corti knocked off the basement mem-

brane by a 400 cycle tone, 125 dcb. above threshold intensity: exposure, one minute; subject, a guinea pig. The lesion is near the helicotrema. "The organ of Corti and the rest of the cochlea appeared disorganized but was not thrown off the basement membrane."

A number of us have heard of the selection of a 4,000 pitch (or one thereabouts) for a landing beam signal for aviators. It seems deplorable to select a signal in this known region of greatest hearing vulnerability. In spite of much correspondence, I have been unable to get either confirmation or denial of the information.

Protection of ears will, of course, safeguard to some degree the aviator — plugs, the helmet and rubber-cupped headphones, all will reduce the outside noise.

One must consider not only sonorous trauma in war and in civil life but also concussions which affect the ear. The ruptured drum with its bleeding is of itself but an insignificant evidence of the damage within, yet it offers a portal of entry for infection that may readily take hold in a congested middle ear. For this reason, douching must be avoided. Petechial hemorrhages anywhere, often, everywhere in the inner ear, in the brain substance and on the meninges, are a common finding at autopsy in war times. Careful testing and accurate diagnosis are impossible in the first few days. Mental dullness, deafness and dizziness may appear as, and if, the patient survives. As to the incidence of tinnitus, there seems to be a difference of opinion. Certainly, mild sonorous trauma frequently causes tinnitus, but when profound deafness comes, I believe tinnitus is less apt to be present because it cannot be heard. For the dizziness, there fortunately supervenes an accommodation to the imbalance. This should occur within a month.

As to immediate treatment, rest is all that is usually suggested. Sedation is rarely required unless brain or meningeal irritation appear. Symptoms of concussion are treated along the well known lines. Massive hemorrhage within the calvarium is not to be overlooked as a possible explanation of symptoms. The same is true of fractures of the petrous pyramid when the patient has been knocked down by an explosion. "Sudden displacement of the brain stem by the impact may

directly injure the VIIIth nerve as it enters the Fallopian canal. Findings similar to those of tumor of the cerebellopontine angle are significant."[5] Dr. Crowe[6] comments: "These ear injuries in some persons result in a series of vague complaints. We can elicit no specific symptoms or signs for the localization of the trouble and, therefore, we have no intelligent therapy to offer." "For some patients, rest in bed for several weeks after a head injury is wise, for others it only increases introspection."

Dr. Collier,[7] reporting on war injuries to the ear in the Spanish Civil War, gives a ratio of 175 injuries by blast, against 33 by bomb fragments. Blast caused rupture of the drum with sharp pain, bleeding from the ear and tinnitus. While the bleeding was on, drainage was assisted by placing the patient on the appropriate side.

Lionel Colledge[7] believes that direct wounds of the temporal bone in war must be fairly common, but many were not recorded, probably because they were often immediately fatal. Negus[7] reports that out of 622 civil air raid casualties, 193 were injuries of the head and neck; 129, craniocerebral; ear cases, 24, or 3.8 per cent.

Where running ears are present, the cases are *a priori* complicated. Chemotherapy is undoubtedly reducing the mortality. Douching should be avoided. Mopping and antiseptic powder insufflation are to be avoided. The deeper parts of the meatus should be left severely alone, at least until serious indications appear.

Most observers argue against immediate operation in wounds of the mastoid process. Fragments of loose bone or metal should be removed, but nothing in the way of extensive operation is advisable. Chemotherapy and the altered circumstances of war, as compared with civil life, have undoubtedly led to this conservative policy.

There is suggested for consideration the evacuation of all running ear cases from the combat zone. This would reduce to some extent the mortality in the 3 to 4 per cent of ear casualties.

Efforts should constantly be made by line and staff officers, as well as by the medical officers, for the more universal use of ear plugs when under fire on the line or during air raids.

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1805 Chestnut Street.

CASSELBERRY AWARD.

The American Laryngological Association has now available a sufficient sum of money to be granted as a prize for a thesis dealing with original investigation and research in the art and science of laryngology and rhinology. Theses or reports of work must be in the hands of the Secretary before March 1, 1942.

For further details address Dr. C. J. Imperatori, Secretary, 108 East 38th street, New York.

MILITARY SYMPOSIUM.

MALINGERING AND NEURO-OTOLOGIC CONSIDERATIONS IN COMBAT SERVICES.*

DR. EUGENE R. LEWIS, Los Angeles.

As I see my assignment in this symposium, it is concerned with practicabilities rather than with exactitudes. Accordingly, I have reviewed past experiences with reference to present and future needs. I propose to indicate in general what offers prospects of proving workable rather than to discuss details and relative merits of various tests.

I spent two years attempting to apply otologic standards to "mass-production" military activities during World War No. 1. This experience compelled much revision of previous notions. Ear tests which had been calculated to meet the demands of military service in many instances served to reject men who were well-qualified; in other instances to pass men who were ill-qualified. Defectives, once duly certified as good military material, cluttered up the service for the duration — and the pension rolls thereafter. Laws of Medes and Persians have nothing on the immutability of army service ratings. A man's hearing defect may be obvious to the man on the street, but if it comes to be rated "normal" in his army record, neither Congress nor the Supreme Court can do much about it. And such things do get into the records. By the same token, good hearing comes to be rated as defective. It is idle to argue that such things must not be allowed to happen; they happen just the same, and with surprising frequency. No amount of high resolve to prevent them in the future suffices to out-promise the promise of their continuing to happen. This may sound pessimistic to the uninitiated, but to those who can recall experiences in active medical service it is just realistic. Blank spaces in paper work are glaring; errors are not. Once paper work has been filled in, attempts to delete errors embalmed therein would bottleneck the progress of a war. Fortunately, the importance of most errors

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tends to shrink as the observer's perspective broadens. Many ill-qualified soldiers have demonstrated the possibility of serving without calamitous results to themselves or to their organizations. What has been thus briefly avowed is not to be understood as deprecating high standards, but as a measure of reassurance against inevitable shortcomings.

Otologic requirements for different arms of military service differ widely. Ear equipment which does not meet the requirements of a pilot, or of a soldier at some listening post, is adequate for other military demands. And no matter how efficient ear equipment may be at one stage of an individual's service, exposure to roaring motors, detonations and other noises incidental to military activity offer constant threat of impairment. Otology cannot do more than confirm the momentary adequacy of cochlear and vestibular function at any stage of an individual soldier's service.

In the presence of satisfactory hearing: deformities of the auricle do not disqualify for military service; chronic otitis externa does not disqualify unless characterized by exacerbations of incapacitating nature; chronic suppurative otitis media is disqualifying except for limited military service; quiescent perforation of the tympanic membrane is not, in itself, disqualifying; retraction of the tympanic membrane is not, in itself, disqualifying; Eustachian obstruction, in itself, is not disqualifying.

Quantitative or qualitative findings of cochlear or vestibular impairment do not, in themselves, constitute evidence of permanence. Such impairments may disqualify for military service only if their permanence be confirmed by repeated findings over sufficient intervals of time.

In evaluating audiograms, cognizance must be taken of wide discrepancies occasionally found between hearing losses indicated by different audiometers; discrepancies of 50 per cent or greater in the case of equally authentic instruments were reported by Beasley to this Society at its last meeting.

Duly established loss of 10 dcb. in the 1,024-2,048 regions is not disqualifying for general military service; requirements for certain special duties must be borne in mind as exceptions.

Let me repeat that audiometric and other precise quantitative and qualitative findings are not, in themselves, proof

of the permanence of any degree of function. Proof requires confirmation by repeated tests at sufficiently long intervals.

Ten to 15 dcb. loss of hearing between 256 d.v. and 3,000 d.v. is not disqualifying for general military service. Coupled with such hearing, as much as 25 dcb. loss in the 128 d.v. region, and as much as 35 or 40 dcb. below 96 d.v. is not disqualifying for general military service. Coupled with such hearing, as much as 25 dcb. loss at 6,000 d.v., and as much as 35 or 40 dcb. loss in the higher regions, is not disqualifying except for special listening device, telephone and radio service. The wear and tear of combat service inevitably threaten to produce vestibulocochlear impairments; most of these are transitory and few are disqualifying for average service demands. There are cases which develop into permanent impairment of some measure; there are others destined to develop similar impairments even under peacetime conditions; and it is manifestly impossible to fortell either the former or the latter. In any event, the *post hoc ergo propter hoc* formula will prevail in accounting for impairment.

Accurate data should be recorded as the only safeguards for all concerned. Fallacies, however, must be expected despite all data. Discrepancies between the facts and pension ratings are not the otologist's business. They are matters between Congress and the individual.

Kipling's "Song of the Banjo" sings the impracticability under war conditions of instruments whose peacetime superiority is admitted:

> "You couldn't pack a Broadwood half a mile —
> You mustn't leave a fiddle in the damp —
> You couldn't raft an organ up the Nile
> And play it in an equatorial swamp."

One is tempted to paraphrase a "Song of the Tuning Fork":

> Audiometers don't fit where caissons roll.
> Graphs to calculate — and complicated masking —
> Soundproofing 'gainst a tumult in the soul!
> How then interpret audiograms? I'm asking.

The audiometer is the instrument of choice for precise measurement of hearing, under suitable conditions — on admission to, or discharge from, service or on investigating special cases remote from war zones. I consider the audiome-

ter impracticable under combat service conditions. The only otologic tests required in war zones are those which can be carried on anywhere by means of voice, whisper, tuning fork, watch-tick, noise apparatus and caloric douche. In selecting tests for combat conditions, precision must yield to practicability.

Malingering assumes widely varying aspects in military service, depending upon circumstance. Under some circumstances it proves to be a relatively trivial matter; under other circumstances a very serious one. Once a soldier has been duly adjudged a malingerer, he becomes subject to courtmartial according to the Articles of War. If courtmartial find him guilty of malingering in order to evade something trivial, the penalty is commensurately trivial. If he be found guilty of malingering in the face of the enemy, the penalty is commensurately severe. Under some conditions, malingering may be punishable by death.

This is not the place for a relative evaluation of methods confirming diagnosis of malingering. When it involves something trivial, due diligence does not require elaborate procedures. When it involves something serious, every available exactitude should be observed before adjudging a man guilty.

It is of vital importance for medical officers to realize fully the nature of their responsibility in establishing a diagnosis of malingering. It is not the function of a courtmartial to adjudge duly established diagnostic evidence; the court's function is to investigate the circumstances and to fix the penalty for guilt. Once the fact of malingering is duly attested, court procedure must take the course provided by the Articles of War.

Diagnosis is the crux of such a trial; and the weight of responsibility resting upon those whose expert opinions establish the fact of malingering before the court cannot be emphasized too strongly.

658 South Bronson Avenue.

MILITARY SYMPOSIUM.

THE EAR IN FLYING.*

DR. JOHN R. POPPEN, Commander (M.C.), U. S. Navy, Washington.

There are three aspects of otology which are of particular interest to Aviation Medicine:

1. The physiology and hygiene of the ventilation of the middle ear.

2. The relationship between flying and the auditory mechanism.

3. The rôle of the labyrinth in aerial equilibration.

An attempt will be made to outline in general and comprehensive terms these three, and what constitutes their particular interest in aviation medicine. Under each heading there will be included certain practical considerations which should serve to focus this interest and point toward methods and means which may be applied to the preservation of flying fitness. You are asked to bear in mind the fact that aviation medicine is almost exclusively concerned with normal physiological processes and hygienic preventive measures and not with those degrees of malfunction and disease which are usually accepted as of clinical significance and requiring the care of a physician. For this reason the following discussion may appear to be out of place in a program otherwise devoted to the clinical practice of otology. This differentiation should not be considered as a separation, however, because in all branches of the healing art there has developed an increasing tendency toward the preservation of health and fitness with a view to reducing the necessity for curing disease. This tendency is the bedrock foundation of aviation medicine and many flight surgeons have experienced the feeling of watching clinical medicine surge in its appointed direction while they rode a somewhat divergent tide of increasing concentration on normal function.

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I.—VENTILATION OF THE MIDDLE EAR.

Inability to preserve equal pressure on both sides of the tympanum is not only a matter of placing uncomfortable stresses upon that organ with varying degrees of distress. Under the amplitude and rapidity of change in atmospheric pressure encountered in modern flying, there result distortion and impairment of hearing and, occasionally, serious pathological changes.

To appreciate the principles involved in this balance of pressure, it is well to review certain anatomic and physiologic considerations.

Barometric pressure is exerted evenly over the entire body. Fluid and solid tissues, being incompressible, are unchanged in volume with changes in barometric pressure. Gaseous contents tend to change in volume to adjust to changes in pressure. The gas contained in the middle ear and its connecting air spaces will tend to expand with decrease in barometric pressure, and vice versa. The only normal compensation for this tendency is the passage of a portion of this changed volume through the Eustachian tube. This is particularly true in case the volume in the middle ear is not increased to compensate for increased barometric pressure. The tendency toward vacuum in the middle ear produces engorgement of the vessels lining the cavities, extravasation and actual hemorrhage. The condition known as aero otitis media is a direct result of this failure to ventilate the middle ear properly.

Gas passes much more readily out of the middle ear than into it. The reason for this is that the Eustachian tube is shaped like two funnels with their apices adjoining. The upper funnel comprises a third of the tube and its walls are osseous. The mucous membrane is in close apposition with the bony funnel, leaving it patent at practically all times. Any tendency for gas to leave the middle ear will find a gaping opening. As a matter of fact, in very rapid experimental exposures to reduced atmospheric pressure the complete absence of any symptoms in the ear suggests that the expanding gas in the middle ear immediately opens the tube, and it remains open during the entire episode.

On the other hand, the lower two-thirds of the Eustachian tube is soft tissue with comparatively flabby mucous membranes over its opening into the pharynx. A demand for ingress of gas into the middle ear meets with a tendency to close the opening on the order of a flutter valve, and increase in the inward pressure tends to exaggerate the condition.

It is almost the rule that descents from altitude require some conscious maneuver to ventilate the middle ear to keep pace with the increasing barometric pressure. Assisting opening of the Eustachian tubes by tensing and relaxing the tensor muscles by moving the mandible will often suffice, but there are a number of individuals who must resort to closing the nostrils and exerting enough pressure in the pharynx to initiate the opening.

The brisk component of this deliberate opening of the tube appears to have practical significance. Many pilots who find it necessary to repeat the procedure during descents at moderate rates will experience no difficulty during the descents made in a high-speed diving attack. In this case, the initial opening is brisk and the tube probably remains open while considerable gas enters the middle ear.

The danger of consciously and deliberately blowing open the tube with the possibility of introducing infectious material into the midde ear has been over-emphasized. It is true that the swelling of the pharyngeal mucous membrane in a "cold" exaggerates the tendency for the tube to be closed, and infectious material is present in abundance, but the amount of air introduced is very small, and gravity, ciliary action and lymphatic drainage all join in returning material introduced in this way into the pharynx. If the danger were great, the incidence of otitis media complicating head colds in aviators would be many times that shown by clinical experience.

Another very significant consideration in replacing gas into the middle ear is the chemical composition of the gas mixture. In equilibrium, the mixture in the middle ear is composed of the same gases and in the same percentage as those in circulation in the capillaries lining the cavity. If the gas introduced has partial pressures in excess of those in the capillaries the excess will be absorbed in the course of time. This produces a reduction in total volume and a greater pres-

sure on the outside of the tympanum. This necessitates the introduction of more gas. This is particularly significant following descents from high altitude during which pure oxygen has been breathed. Behnke has described cases in which high concentrations of oxygen have been absorbed during sleep following such descents, producing complete filling of the middle ear by serum and hemorrhage. He recommends that only ambient air of 21 per cent oxygen be introduced during the last stages of the descent — from 10,000 feet.

This is an extreme degree when air of normal composition is introduced during the entire flight. It is quite common for passengers in commercial air liners to complain that the pressure will not equalize for several hours after descent. This is because the 21 per cent of oxygen must be absorbed down to 12 to 15 per cent in the capillaries. Happily, there is a simple solution which requires very little attention. If the air introduced is alveolar air, it will approximate very closely the composition of that in the capillaries. Thus, if the ear is inflated at the end of expiration following moderate breath-holding, the period of recovery will be very materially shortened. This simple procedure is offered as a therapeutic means to those who have experienced protracted delay in re-establishing normal pressure in the middle ear.

Another important consideration in connection with the chemical composition of the gas in the middle ear is the fact that it explains the disheartening results of attempting to ventilate the ear by using exotic gases. On the assumption that the greater coefficient of permeability of helium will permit more facile introduction of this gas into the middle ear, there have been a number of experiments using this gas alone or in combination with oxygen. Of course, the introduction of gas is a matter of the passage of bubbles rather than permeation, but, assuming that the helium will penetrate more rapidly than nitrogen, it will have to be absorbed. Equilibrium cannot be established until the composition of gas in the middle ear is the same as that in the capillaries. So the introduction of helium is only a stop-gap and oxygen and nitrogen in proper combination will have to be introduced eventually.

This subject should not be dismissed without reference to the hygiene of the nasopharynx in this connection. Correct

ventilation of the middle ear is impaired by any condition which produces swelling of the pharyngeal mucous membrane. Of course, gross pathology should be adequately treated. Conditions of this nature are a cause for rejection in military aviators. Transient and minor swelling incident to a "cold" can be shrunk by topical applications or the inhalation of benzedrine.

II. – THE RELATIONSHIP BETWEEN FLYING AND THE AUDITORY MECHANISM.

The impairment of hearing incident to the changes in pressure in the middle ear is of little significance and lasts only as long as unequal pressure prevails. Sclerosed ossicles may have a reluctance to return to normal positions, but the existing impairment in hearing is very little exaggerated by this circumstance. A few years ago, flying was advanced as pretty much of a panacea for such types of deafness. It must be assumed that this therapeutic procedure was advocated in those cases in which massage was indicated. This is the only conceivable type of deafness which could be relieved by flights in airplanes. If so, flying would come off a very poor second in competition with any approved office equipment.

The question of permanent impairment of hearing or damage to the auditory mechanism by continued flying is of less concern than the more current fatiguing effects of the noises encountered in aircraft. A number of surveys have been conducted to determine the incidence and degree of impairment in pilots who have flown for a number of years. When these surveys have been carefully controlled to rule out the effects of very recent exposure, unsatisfactory ventilation and disease, they show a fairly high incidence of reduction of hearing in the high frequency range of the audible spectrum. But, in turn, when these audiograms are compared with similar findings in the same age group who have not flown, it is generally found that flying has not been a considerable contributory factor. Hearing is reduced for considerable periods after flight, but this is evidence of fatigue and does not mean permanent or prolonged impairment.

In contemplating the fatiguing effect of noise, we are immediately faced with the inadequacy of existing definitions of

fatigue. The word is quite generally understood in its broader implications as exemplified in the subjective feelings and objective findings following loss of rest and strenuous exertion. There are many data on the chemical changes incident to exhausting treadmill studies. There are laws about refractory periods and reduced conduction of neurones and nerve fibres. But in evaluating the relative significance of physiological, chemical, neural and psychological findings and the weighting of end-points in examination methods with a view to measuring the total effect upon the entire organism, there is still much to be desired. This is particularly true when applied to a study so apparently abstruse as the determination of the fatiguing effect of noise.

There is very little information on the effects of noises which involve the entire audible spectrum, or particular sounds which include comparatively narrow bands in the lower or higher frequencies. The comparative significance of vibrations within or beyond audible ranges, critical intensities, individual variations and many other detailed findings is of extreme importance to the study of the problem. It is characteristic of the response of science to our present emergency that there has been set up in recent months a comprehensive and well planned study of precisely these points, and there is great promise that within a short time we will have many of the answers.

The detailed answers are of extreme importance in directing studies of means to protect aircraft personnel from the effects of noise and vibration. This will be appreciated after consideration of some of the principles involved. A preponderance of noise in an airplane emanates from the propeller tips, the explosion cycle of the motor and the operation of gears. These are of low pitch but high intensity. High-pitched sounds result from aerodynamic sources such as whistles through leaking joints in hoods, ventilating systems, etc. The design and construction of aircraft and accessories with a view toward appropriate absorption of or insulation against sounds of different frequencies involves different principles. Bearing in mind the fundamental requirements of weight, space, structural configurations, serviceability and cost, it makes a lot of difference whether protection shall be directed toward high or low tones and the decision must be

based upon the relative fatiguing and disturbing capabilities of the two. These questions are the subject of extended and accelerated study at the present moment, and there is promise of early solution to many of them.

The effect upon hearing of reduced atmospheric pressure and anoxia, the development of microphones and receivers more adapted to service use, the feasibility of a chest type microphone, the optimal pitch for radio tones, etc., are among the questions on which accelerated study is being applied.

III. – THE ROLE OF THE LABYRINTH IN AERIAL EQUILIBRATION.

The labyrinth has held a conspicuous place in the minds of those interested in aerial equilibration since the beginning of aviation medicine. Its position has been altered, to be sure, but it continues to be of tremendous interest partly because it has been responsible for the development of considerable controversy.

From the point of view of the established importance of labyrinthian functions in the maintenance of position in space in terrestrial existence, and particularly the intimate association between its dysfunction and disequilibrium, it is only natural that it should have been accredited an outstanding rôle when the drama of flying equilibrium was first written. Add to this the obvious necessity of maintaining equilibrium in a three-dimensional environment with intricate and profound gyrational components which were known to be the special forte of this organ, and we have the groundwork for assumptions which were so deeply rooted that they tended to becloud the issue. It is significant that many of these assumptions were strong enough to delay a more rational interpretation of events and functions on the basis of more exact and fundamental principles.

A more accurate estimate of the rôle of the labyrinth is made apparent when the problem is approached from the point of view of the physics of flying in airplanes and the direction and forces of gravitational changes incident thereto. There are features of these forces which are peculiar to the movement of aircraft through the air and are of such importance that they control the relative significance of the functions of equilibrium and orientation.

The most important consideration in this connection is the fact that a flying airplane assumes, to all intents and purposes, the status of a celestial body. This is because it engenders within itself gravitational forces of such a nature and degree that they virtually deny those of the earth. In even the simplest maneuver the direction of gravitational force remains normal in direction, but greater than normal in intensity because they must overcome the normal pull of gravity. This is essential to preserve aerodynamic stability.

The first essential in aerial equilibration is the preservation of a satisfactory relationship between the pilot and his airplane. This is made possible by the preservation of his proprioceptive somatic senses as included in the group of touch, deep and superficial pressure sense, muscle and tendon sense, kinaesthesia, etc. Disregarding, for purposes of properly focusing attention, the intimately close relationship between these senses, the labyrinth, the cerebellum, the eyes and all other associations, the significant consideration for the pilot is the fact that the physical forces upon which he must rely for this preservation are those which constitute the correct and adequate stimulus for the somatic proprioceptive senses. To preserve a satisfactory relationship to his airplane, he must rely on those afferent impulses which pass up the posterior column of the spinal cord. It has become axiomatic that a pilot gets his "feel of the air" through the seat of his pants.

This fact has been repeatedly demonstrated under circumstances in which rigid mechanical contact has been interrupted, either deliberately or inadvertently. In a recent experimental setup it was desired to interpose a layer of fluid between the body and the seat, and the fluid was designed to approximate the specific gravity of blood. The disruption of continuous solid contact with the airplane produced by this situation was completely and alarmingly intolerable to the experienced pilot. It deprived him of his "feel of the air."

When the gravitational forces developed in an airplane are of such a nature and degree that the adequate stimulus for the labyrinth predominates over that for the somatic senses, we find ourselves in rather distressing circumstances. So long as the airplane progresses through moderate turns and

spirals, without slips or skids, under perfect control and with the air passing over the airfoils and control surfaces in conformity with sound aerodynamic principles, the gravitational changes reach the body in the form of pressure of varying intensity, but always along normal lines. It is the somatic senses which perceive these changes and translate them into reflexes and information of use in preserving equilibrium. But when the airplane executes evolutions in which the forces attain to such direction and amplitude that they produce a preponderant stimulation of the labyrinth, it is cutting lines of gravitational forces in extreme and abnormal fashion. Such preponderant stimulation, which will overpower the more normal somatic stimulation, results from rotational movements of comparatively short radius. These are encountered in such spectacular maneuvers as snap rolls, prolonged tight turns and spins.

It is important to note that the labyrinthian stimulation under these abnormal circumstances, through its concomitant vertigo and postrotatory nystagmus, ultimately reaches the high centres and consciousness in strong concentration, and the information it imparts is always wrong.

Thus, the rôle of the labyrinth in aerial equilibration becomes a secondary one. To preserve to the pilot a satisfactory adjustment to his airplane, the labyrinth must not detract from the leading rôle of the somatic senses. It has been advanced that it might be better for flying if the labyrinth could not be easily stimulated. Certainly, a hypoirritable labyrinth is more to be desired than a positive Romberg, and a hyperirritable labyrinth may be a positive danger.

There is one redeeming feature in the fact that the preservation of equilibrium between the pilot and his airplane is only a part of the picture. In many respects, it is much more important to preserve orientation with the earth's surface, and since this can only be accomplished by vision, it is possible for the pilot to exercise his experience and reliance upon his instruments, keep his labyrinths in their proper rôle and return safely to Mother Earth.

HONOR WHERE IT IS DUE.

The Kansas City Society of Ophthalmology and Otolaryngology recently honored one of its own members when it presented a gold medal to Dr. J. L. Myers in appreciation of his service in all of its activities, especially in promptly and cheerfully aiding its advancements in every way. Anyone familiar with the activities of this society knows how richly Dr. Myers merited such recognition. Few men in any organization show the devotion to service which he has maintained toward this society. He has given of himself to the program of this energetic group. Appreciation of his spontaneous energies is not confined to this society, of which he has been a leader for years, but also the American Academy, as well as the Kansas City community in general recognize and appreciate what this veteran has done and is doing to promptly and efficiently discharge all duties assigned to him.

He has been engaged in practice in Kansas City since 1916 and has been Secretary of the Otolaryngologic Department of the American Academy of Ophthalmology and Otolaryngology since 1920.

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